A SSESSMENT OF THE INTRATESTICULAR RESISTIVE INDEX BY COLOUR DOPPLER ULTRASONOGRAPHY MEASURMENT AS A PREDICTOR OF SPERMATOGENESIS.

Dissertation submitted to

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M.Ch (UROLOGY) – BRANCH – IV

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DECLARATION

I solemnly declare that this dissertation titled **"ASSESSMENT OF THE INTRATESTICULAR RESISTIVE INDEX BY COLOUR DOPPLER ULTRASONOGRAPHY MEASURMENT AS A PREDICTOR OF SPERMATOGENESIS"** was prepared by me in the Department of Urology, Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai under the guidance and able supervision of **Prof.R.Jeyaraman MS, M.Ch.,** Professor & Head of the Department, Department of Urology, Madras Medical College & Rajiv Gandhi Government General Hospital, Chennai. This dissertation is submitted to the Tamil Nadu Dr. MGR Medical University, Chennai in partial fulfillment of the university requirements for the award of the degree of M.Ch. Urology.

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Place: Chennai
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CERTIFICATE

This is to certify that the dissertation titled **"ASSESSMENT OF THE INTRATESTICULAR RESISTIVE INDEX BY COLOUR DOPPLER ULTRASONOGRAPHY MEASURMENT AS A PREDICTOR OF SPERMATOGENESIS"** submitted by **Dr.Ahmed Marzook.S** appearing for M.Ch. (Urology) degree examination in August 2013, is a bonafide record of work done by him under my guidance and supervision in partial fulfilment of requirement of the Tamil Nadu Dr.M.G.R.Medical University, Chennai. I forward this to the Tamil Nadu Dr.M.G.R.Medical University, Chennai.

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INDEX

ABBREVIATIONS

CDUS	:	COLOUR DOPPLER ULTRASONOGRAM
PSV	:	PEAK SYSTOLIC VELOCITY
EDV	:	END DIASTOLIC VELOCITY
RI	:	RESISTIVE INDEX
PI	:	PULSATILITY INDEX
OAS	:	OLIGOAESTHENOZOOSPERMIA
iOAT	:	IDIOPATHIC OLIGOASTHENOTERATOZOOSPERMIA
FSH	:	FOLLICLE STIMULATING HORMONE
LH	:	LUTEINIZING HORMONE
TRUS	:	TRANSRECTAL ULTRASOUND
GnRH	:	Gonadotrophin Releasing Hormone
CRH	:	Corticotrophin Releasing Hormone
GHRH	:	Growth Hormone Releasing Hormone

INTRODUCTION

As we have entered the twenty-first century, a number of medical, environmental and social changes have profoundly affected human reproduction. The ever expanding industrialization and the growth of fabricated materials pollute our surroundings in unpredicted ways with possibly distressing effects on human health and fertility.

Infertility affects 14% of the reproductive group of the world's population. The disturbance in the males count to 20% of infertile couples and 26% when both partners are combined. Though the modern technology may provide solace to couples seeking treatment for infertility including the use of donated gametes, the socio-economical and political hardships surrounding the available resources have deterred the advancement not only in the developing world but also in the West.

The couple are advised multiple investigations and they undergo lot of blood tests and imaging studies often. Especially, the male gets stressed to evaluate his semen analyzed time and again. Hence the tool to assess spermatogenesis as and when needed, gets better if it could be done non-invasively and easily. The concept of

Color Doppler Ultrasound in quick evaluation of the Resistive index in the testicular arteries throws light on the assessment of spermatogenetic potential of the subfertile male in the modern world especially in the followup.

The testes generate the gametes by the process of spermatogenesis. The source of vascular supply to testis is from internal spermatic arteries which form the anastomosis with artery to vas deferens and cremastric arteries. Stable vascular supply is needed for the function and maturation of the gonads. Adequate vascularization of the testes is required for two main functions, namely transport and mobilization of endocrine factors and metabolites, as well as homeostasis of temperature within the gonad**.** Blood flow is controlled by two factors when flowing through a blood vessel across a fixed point, i.e. vascular resistance and its pressure, which is expressed as[1]:

BLOOD FLOW = PRESSURE/RESISTANCE.

Colour Doppler UltraSound (CDUS) is one of the fastest and most trustworthy methods of calculating the blood flow, clubbing the data related to both the anatomy and the flow velocity, thereby giving a rapid assessment in formal studies.[2] But, CDUS is mainly

used for assessing the vascularity in various clinical entities like varicocele, torsion testis, infective pathology and neoplastic conditions in day to day practice. The assessment of peak systolic velocity(PSV),end diastolic velocity(EDV) and the mean velocity are used to define various indices like Pulsatility index (PI) and Resistive index(RI).

Resistive index has been used to define intratesticular blood flow. Elevated RI & PI of capsular branches of testicular arteries on unenhanced CDUS examination may give a clue to the defective microcirculation in the gonad. Elevated RI indicates ischaemia and indirectly that of decreased spermatogenesis too as the latter depends on the blood supply. Pulsatility and flow resistance may be gauged qualitatively, either by visual inspection of the Doppler spectrum waveforms or by listening to the auditory output of Doppler instrument.

There are multiple studies, showing the relationship of the arterial impedance in testis with the spermatogenesis. The pioneer work was done by Biaigotti[3] et al where he had calculated the sperm production rate score (SPRS)as:

$$SPRS = \frac{C \times V \times M/100 \times Mn/100}{D \times TV}$$

Where C= sperm concentration,

V= volume of the ejaculate,

M=Type A motility

Mn=Morphologically normal,

D=days of abstinence and

TV=Bilateral testis volume

He was able to demonstrate the various causes of dyspermia related to the various parameters like PSV, EDV and RI while comparing with FSH levels and volume of the testes. RI and especially PSV could also differentiate the obstructive and non-obstructive causes of azoospermia. RI assumed more importance because the angle dependent technique used in evaluating PSV and EDV is eliminated when the former is used as the predictor. This present study aims to illustrate that abnormal sperm count has a strong association with the resistive index of the intratesticular arteries. .

AIM AND OBJECTIVE

The aim of the present study is to investigate the value of the Resistive index (RI) of intratesticular arteries and to establish diagnostic criteria for normal and pathological sperm counts on the basis of colour doppler USG.

REVIEW OF LITERATURE

FUNCTIONAL ORGANISATION OF TESTIS:[4]

The testes generate the sex gametes of the male and the related hormones namely the androgens. Spermatogenesis is defined by the process connected with the generation of gametes, whereas steroidogenesis is defined by the chemical reactions mediated by specific enzymes resulting in the release of steroid hormones in men. Both these processes happen in separate compartments which are unique in both morphology and function .The tubular compartment which consists of the seminiferous tubules and the interstitial compartment between them. (fig:1)

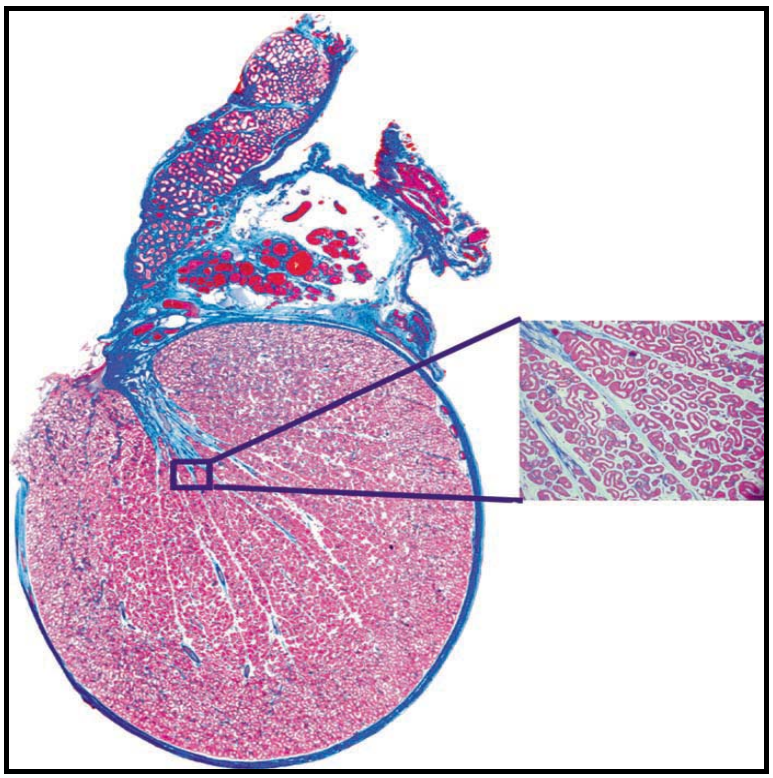


Fig 1: Histological architecture of testis with compartments

Both compartments are closely related to each other though they are anatomically separate. The coordinated function of these two compartments is vital for the normal production of sperm, both in terms of quality and quantity. Endocrine regulation means the hypothalamus and the pituitary gland govern the function of the testis and its compartments. But it is the paracrine and autocrine factors ,i.e, the local control mechanisms that mediate and modulate these endocrine effects at the testicular level.

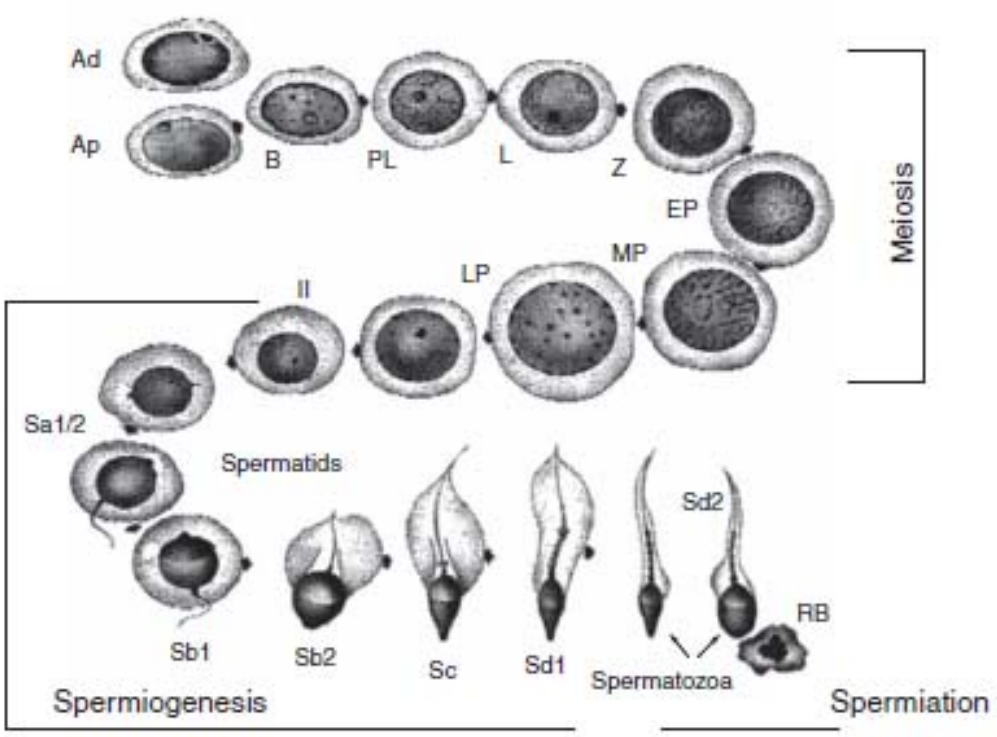


Fig 2: Schematic representation of all germ cell types that occur in the human seminiferous epithelium. Ap spermatogonia enter the spermatogenic process (arrow on the cell indicates direction of germ cell development). Ad spermatogonia are believed to constitute the testicular stem cells. Ad = A-dark spermatogonium, Ap = A-pale spermatogonium, B = B spermatogonium, Pl = preleptotene spermatocytes, L = leptotene spermatocytes, EP = early pachytene spermatocytes, MP = mid pachytene spermatocytes, LP = late pachytene spermatocytes, II = 2^{nd} meiotic division, RB = residual body, Sa1 – Sd2 = developmental stages of spermatid maturation

Spermatogenesis begins with the maturation of primary germ cells,namely the spermatogonia and finishes with the production of a fully mature sperms called the spermatozoa. The production of sperms involves four phases (fig: 2 &3).

1) **Spermatogoniogenesis**: Multiplication and distinction of spermatogonia (diploid) by mitotic division.

2) Division of spermatocytes (tetraploid) by meiosis into spermatids (haploid).

3) **Spermiogenesis:** Biotransformation of spermatids into sperm.

4) **Spermiation:** Exit of sperm from the epithelium in the germ cell into the lumen of seminiferous tubules.

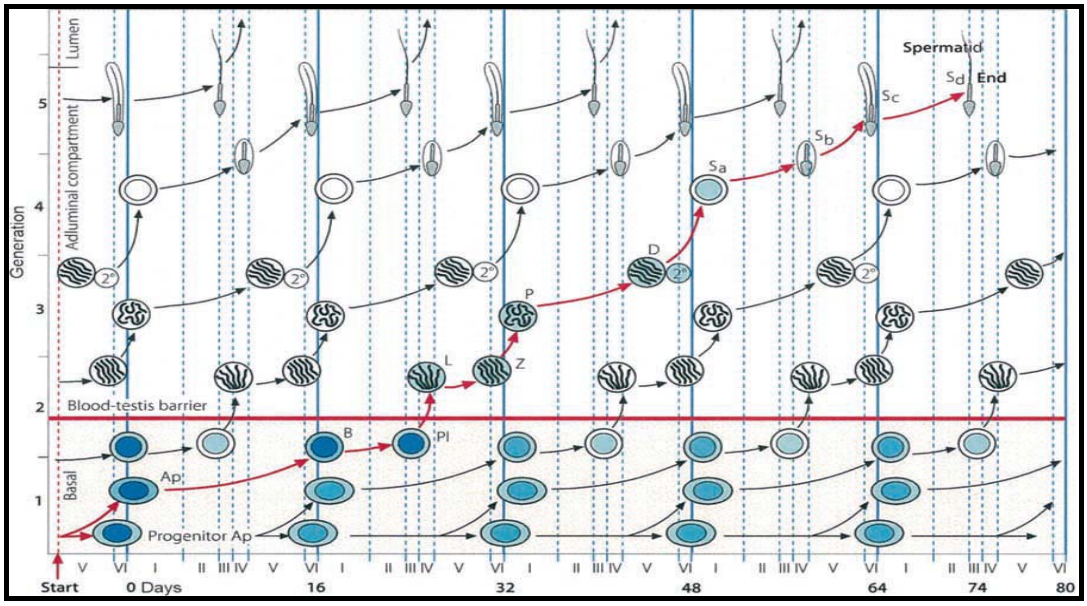


Fig 3: The architecture of seminiferous epithelium shown schematically

BLOOD SUPPLY OF THE TESTES:[6]

The testis primarily receives its blood supply from the gonadal ateries, also referred to as internal spermatic arteries via the spermatic cord to testes, through the inguinal canal and they form extensive anastamosis with cremasteric artery and arteries to vas deferens.

Adequate blood supply to testis is considered vital for normal spermatogenesis.

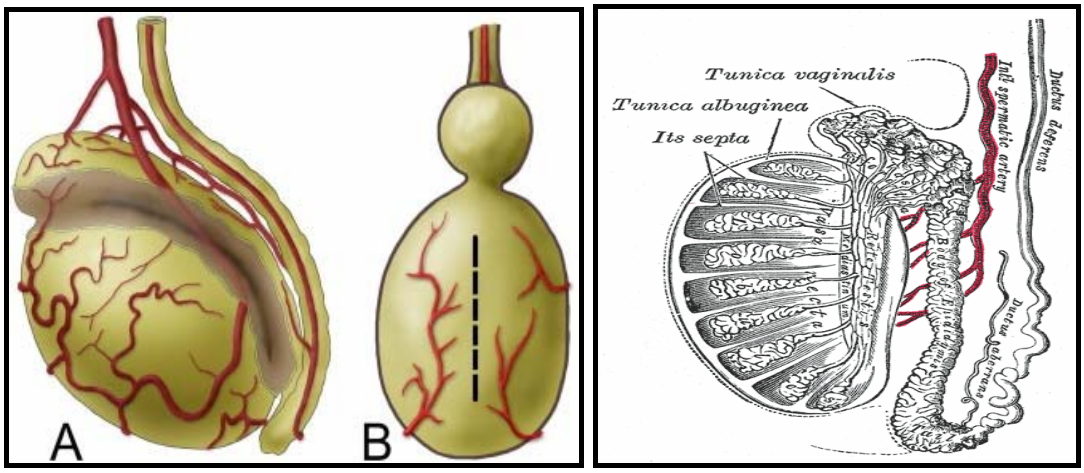


Fig: 4-Arterial supply of testis and the testicular architecture

The testicular artery (also referred to as the internal spermatic arteries by the medical diaspora earlier) is a paired branch of the abdominal aorta,so there is an artery for each gonad.(fig: 4)

They are two small arteries of variable length, and they branch from the ventral aspect of the abdominal aorta slightly distal to the arteries to the kidney.

Both the testicular arteries take a oblique course inferiorly and later appear in the retroperitoneum, over the surface of psoas major muscle. The right testicular artery lies ventral to the inferior vena cava and dorsal to the middle colic vessels, and the distal part of the ileum and its blood supply namely the ileocolic artery. The left testicular artery lies posterior to the the descending colon, arteries to the same bowel and the artery to sigmoid colon.

Both the testicular arteries pass over the ureters crossing them obliquely and the distal part of the external iliac vessels to enter the deep ring of the inguinal canal, and passes through the ring and then along the canal, with the other contents of the cord to reach the scrotum.At that anatomical location, it becomes tortuous, and terminates into multiple branches.

Two or three of the branches of testicular artery travel along the vas deferens and the epididymis supplying the latter and then undergo anastamoses with the artery to the vas. Other branches penetrate the posterior aspect of the outer tunical covering, giving rise to capsular arteries, and then supply the parenchyma.

The gonadal artery gives rise to few tiny branches to the ureter, and few small branches to the cremaster as it passes

along the canal of the inguinal region. The testicular artery has one to three branches of different calibers (most commonly upper and lower pole) that exhibited four different patterns[7] in the testicular corrosion cast study (1) 69.7%, two main terminal branches; upper polar (segmental) branch directed toward the upper anterior aspect of the testis and a lower polar (segmental) branch directed toward its lower posterior aspect. In 80% of this pattern the lower polar branch gave branches anastamosing with branches from the cremasteric artery. In 20%, each upper and lower polar branch anastamosed with the cremasteric artery [2] 15.8%, the upper polar branch, middle segmental branch near the middle of the mediastinum testis then continued to the lower end of the testis supplying it and the epididymis then it recoursed upward and forward [3] 7.9%, it descended along the mediastinum testis giving three extratesticular terminal branches: upper polar or segmental, middle segmental, and lower polar or segmental branches; each gave many centripetal branches supplying the testis from behind forward. [4]6.6%, it gave out an upper polar branch before reaching the upper pole of the testis then continued along the mediastinum testis to reach the lower end of the testis and recoursed forward and upward to supply its anterolateral part. The most terminal part of

the testicular artery anastamosed with branches from the cremasteric artery.

In other cases, the artery did not divide (5.0%), descending straight or wavy along the mediastinum testis without extratesticular terminal branches. Many centripetal branches originated directly from it to supply the testis, epididymis, and vas deferens.

The testis requires adequate and reliable vascular supply for its basic physiologic functions and development. The blood vessels to testis serve two important functions namely transport and distribution of hormonal factors and their metabolites and maintenance of optimal temperature. The vascular supply of the testicular parenchyma by means of the centripetal arteries is coherent with the lobular segments of the seminiferous tubules. Each lobular segment is supplied with a corresponding branch from which segmental arteries arise sequentially which supply blood to the lateral parts of each lobule. The latter give rise to capillaries that become branched and thus end up dividing the Leydig cells and ultimately form the venous drainage.

A decrement in the vascular supply of the gonad leads to ischaemic insult. To provide scientific data, a quasi-experimental analysis was conducted to evaluate the effect of the impediment of growth of the testicular artery by surgical means on testicular volume and the testicular histological architecture in bullocks.[8] Surgical control of the development of the testicular artery was performed one side in young bullocks and their effect on testicular development was analysed. Histological comparison between the experimental testis and hypoplastic testis was performed. The growth rate of testis, the mean testicular volume and the average diameter of the seminiferous tubules from the experimental side were significantly smaller in the artery-restricted and hypoplastic testis. In the sham-operated testis, the testicular parenchyma is significantly replaced by interstitial tissue and spermatogenesis was absent in most if not all the seminiferous tubules. It is therefore recommended that the artery-restricted testis might be used as a model for analysis of testicular hypoplasia in vascular compromised situations.

OBJECTIVE SEMEN ANALYSIS[4]

In clinical andrological practice, the examination of the ejaculated material is considered to play a significant role in the

evaluation of infertility by protocol. A semen sample is produced by means of masturbation after 2-5 days of abstinence for its analysis. The abstinence is just for standardization so that samples from different men can be compared.

But, various andrological studies have emphasised that the measurement of sperm concentration, sperm motility and the morphology of spermatozoa is affected by subjective variables predominantly. Semen analysis is now being standardized according to WHO laboratory manual, 2010 across the globe.[9]

The standard tests are the macroscopic evaluation in the form of appearance, volume and pH. The microscopic tests are the observation for aggregation, agglutination and non-sperm cells. Mixed antiglobulin reaction test is needed if agglutination is profuse and leucocyte peroxidase activity test is done when non-sperm cells are found in plenty. Apart from this, sperm motility, vitality if the motility factor is low, the sperm concentration and morphology are the basic evaluation.

The motility factor ensures objective data on velocity. Along with measuring the population of motile spermatozoa, these techniques allow calculations of kinesmatic variables, such as

velocity and linearity of the sperm movement, the amplitude of displacement of the sperm head laterally and frequency of the beat of the sperm head.

The optional tests are to find indices of teratozoospermia,pan-leucocyte CD4 staining,biochemical values of semen and in-vitro mucus penetration tests.

ENDOCRINE FACTORS[5]

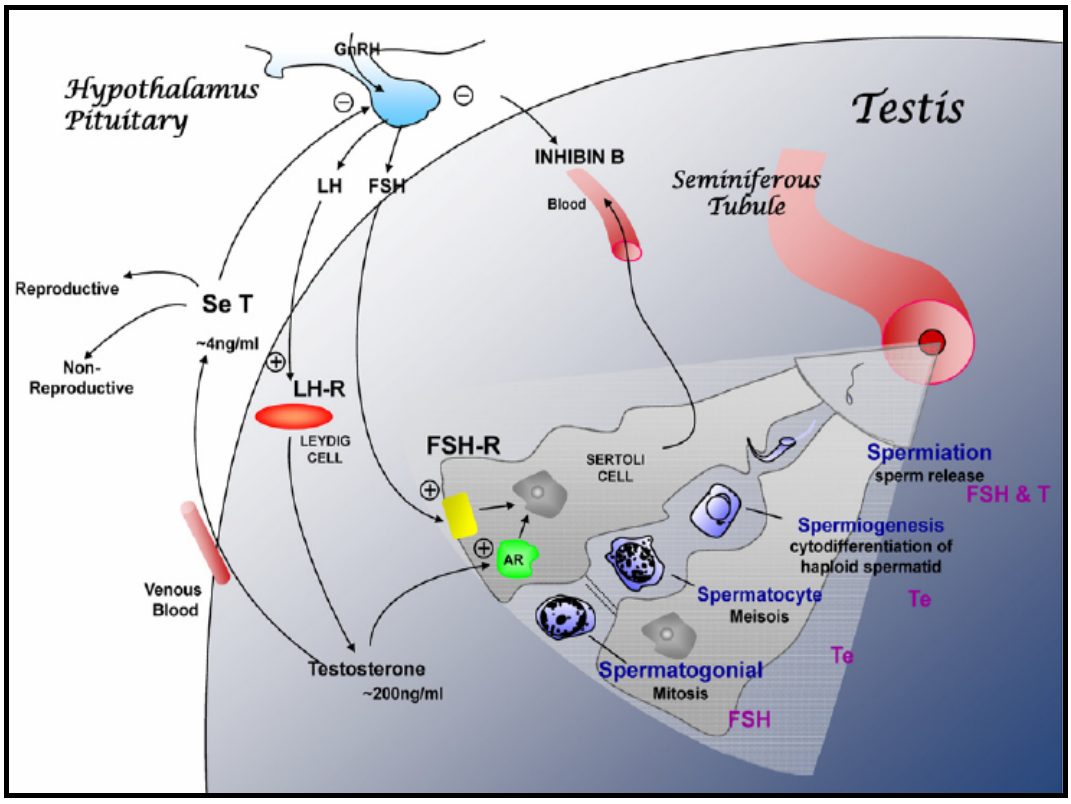


Fig:5- Schematic representation of endocrine regulation of spermatogenesis

Hypothalamus and hypophysis regulate the important role of the testisi.e, androgen production and gamete maturation. This is achieved through the gonadotropin releasing hormone (GnRH)

secreted by the hypothalamus and gonadotropins secreted by the hypophysis (fig:5).

Significantly, this loop between hypothalamus and pituitary is regulated by a negative feedback mechanism effected by factors secreted by testes. The release of Luteinising Hormone and Follicle Stimulating Hormone is suppressed by testosterone. FSH secretion is regulated by the protein hormone inhibin B.

The endocrine effects on the regulation of sperm production is performed by stepwise initiation, maintenance, reinitiation by which a full cycle of sperm production starts in the pubertal age, maintained by the hormones and there is re-stimulation of the production of gametes after a brief pause and this is regulated by testosterone or FSH alone, but only to a qualitative extent which means sperm production is subnormal in spite of presence of all germ cells. It is FSH, which kindles the permanent maturation in the sertoli cell and the seminiferous epithelilum when the pubertal age occurs. Quantitative improvement where all germ cells are in normal numbers, in physiological conditions, require both LH and FSH. The collective action of gonadotropins and testosterone is essential for the three phases of normal spermatogenesis. Men with severe azoospermia or severe oligospermia with FSH levels of >10

IU/L is indicative of non-obstructive azoospermia or testicular dysfunction. If the level of FSH is in the low normal range, it may indicate extrinsic duct obstruction leading to low levels of sperm or its absence. FSH levels <1.5 IU/L indicate hypogonadotropic hypogonadism.

In addition to the above said factors, there are also local regulators like immunological factors, growth and stem cell factors, hypothalamic factors like GnRH, CRH, GHRH, ceruloplasmin, dynorphins, oxytocin and vasopressin.

Blood flow is controlled by two factors when flowing through a blood vessel across a fixed segment, i.e. vascular resistance and its pressure, which is expressed as:

BLOOD FLOW = PRESSURE/RESISTANCE

CDUS (Colour Doppler ultrasound) is one of the most consistent, easily reproducible, convenient and swift techniques of assessing the flow of blood. It combines anatomical representation along with velocity measurements, and thus enables a quick evaluation in regular clinical practice.[1,10] This can be used to identify testicular neoplasms where vascularity is very evident and the same phenomenon is also used to differentiate from the avascular cysts,

inflammatory abscesses and haematoceles. Spectral doppler, in particular has proved its use in demonstrating low resistance flow within tumor vessels. Increased blood flow has been found in infective pathology like epididymitis and orchitis also, clearly visualized on CDUS. The prime use of testicular CDUS is confirming the diagnosis of varicocele where numerous serpiginous veins of unusually larger caliber are found along the epididymis and spermatic cord. Most importantly, asking the patient to do a Valsalva maneuver, the reflex within the veins are also investigated in varicocoele. In emergencies like testicular torsion, CDUS plays a pivot role ,proving 86 to 100 % sensitive and 100% specific for diagnosis and the salvagablity of the pathological testis where in differences in perfusion is easily made out.

Doppler indices have been routinely calculated to evaluate blood flow and impedance to vascular flow that cannot be assimilated from velocity data as a single measurement. These indices depend on the assessment of PSV, EDV and the mean velocity. Commonly employed indices are the pulsatility index (PI) along with RI (resistive index). The RI has been effective in assessment of intratesticular blood flow, this index has been useful in humans as in the animals. With Color Doppler, both centripetal

and centrifugal arteries are seen as short vessels or simple color dots. Trans-testicular, otherwise known as trans-mediastinal arteries are appreciated in as many as 50% of the people subjected to examination, as vessels of larger diameter and length that pass through the testicular parenchymatous zone, they connect the subcapsular space with the mediastinum testis. Trans-testicular arteries are noted in few as asymmetric and are seen, more commonly,in the upper part of the testicular parenchyma. Approximately 50% of trans-mediastinal arteries, accompany a vein. The visualized epididymal views show almost no vascular activity on color doppler.

Elevated Resistive index and Pulsatility index of the branches of testicular arteries over the tunical capsule on unenhanced CDUS investigation could be a sign of sub-optimal testicular microcirculation in patient with varicocele on clinical examination ,and arterial blood flow in the testis was noted to be considerably decreased in males with varicocele ,this may reflect the sub-optimal testicular microcirculation.

Consequent of impaired arterial blood supply to the testis, defective spermatogenesis can be due to impaired metabolic energy in the microvessel bed of testicular parenchyma.[11]

Another significant factor that is offered as an explanation to the positive pathophysiological correlation between PSV, EDV, intratesticular RI and spermatogenesis are the androgens that act on the testicular vessels and as it has been appreciated in the testes of the subfertile men, the layer of lamina propria in the seminiferous tubules show histologic evidence of 'hyalinization' and more frequently is visualized by similar changes within the vessel walls of testicular arteries. Testicular arterioles reveal enlarged endothelial cells, abundant adventitial tissue in the sub-endothelial layer with a significantly narrow lumen on electron microscopy in infertile men. Analysis of histologic data demonstrated a noteworthy increase in the surface of intimal and adventitial layers of arterioles and medial layer of venules. Hence, it was concluded that hyalinization affects both testicular arterioles and venules. This pointed to the fact that the luminal patterns of the testicular vessels, arteries in particular, correlate with sperm production.[12]

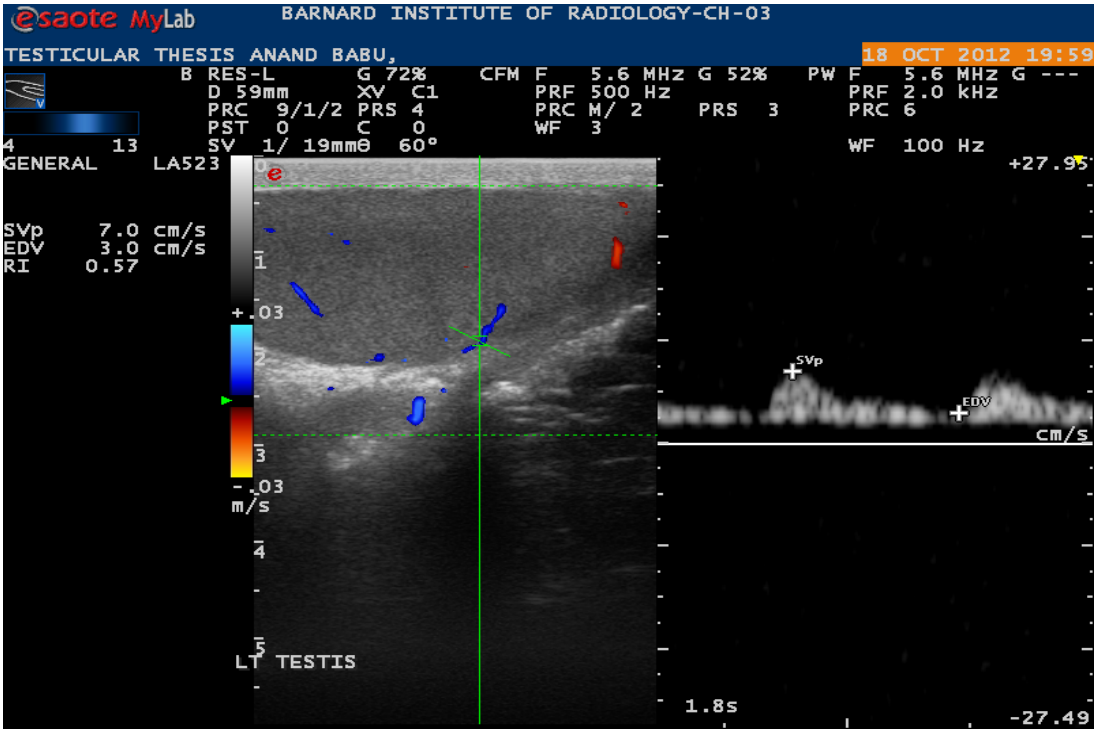


Fig-6: CDUS showing measurement of PSV, EDV and RI

Pulsatility and flow resistance may be gauged qualitatively, either by visual inspection of the Doppler spectrum waveforms or by listening to the auditory output of doppler instrument. Qualitative assessment of pulsatility is often sufficient in most vascular diagnosis. But in certain situations like assessment of renal transplant rejection or measurement of vascular resistance in testes, quantitative measurement of pulsatility becomes mandatory and also desirable. The most commonly used measurements are the pulsatility index of Gosling, the resistive index of Pourcelot-the systolic/diastolic ratio.(Fig:7)

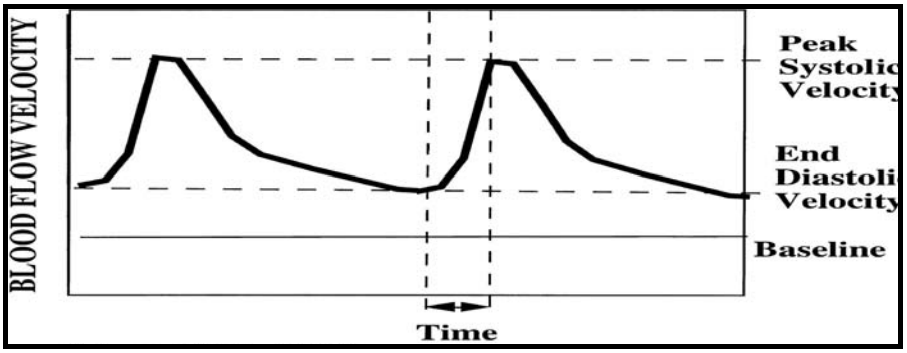


Fig :7 showing the calculation of PI and RI

$$\textbf{RI} = \frac{\textbf{PSV-EDV}}{\textbf{PSV}} \quad \text{and} \quad \textbf{PI} = \frac{\textbf{PSV-EDV}}{\textbf{MV}}$$

Pourcelot described the "Resistance Index" employed in the evaluation of doppler velocity waveforms (PSV-EDV/PSV), reflecting the vascular resistance distal to the point of doppler sampling. Pourcelot has been attributed to have made seminal contribution on color-coded Doppler images. The RI is a misnomer and should originally be called the "impedance index" because resistance and compliance interact to alter the Doppler arterial waveform. Vascular compliance which is defined as the change in volume of a vessel with a change in pressure, also plays a crucial role in affecting the RI. [13]

Gumbsch[14] et al. and M kutzler[15] et al used CDUS to demonstrate that the RI values in the intratesticular arteries could help in characterisation between the fertile and infertile animals that included 42 dogs and 14 camelids respectively in spite of

their normal clinical examination, and also by vascular integrity as suggested by CDUS.[13] Tarhan[16] et al conclusively demonstrated that torsion of the testis on one side does not decrease the blood flow on the opposite side, on colour doppler, in his study on 14 dogs. Pozor and McDonnell[17] objectively demonstrated the use of CDUS in assessing the testicular blood flow in stallions, and to conclude standard measures. Equitable results were obtained for right and left testis respectively.

Such experimental studies demonstrated without doubt the significance of CDUS in identifying the testicular blood supply.

Atilla[18] et al measured arterial impedance and testicular diameter employing the CDUS on 22 adults with unilateral cryptorchidism and used the arterial impedance of the opposite normal testes as controls. After that inguinal orchiectomy was perfomed in all patients and histological examination of the testes were done. The study revealed that the RI of the testicular artery using CDUS which depicted the vascular impedance provides better information on the histology than the volume. The testicular long axis and peak systolic velocity measured by CDUS may be informative tools for the prediction of spermatogenesis. Eytan et al in his study on the intratesticular RI division, has stressed about the

association of intratesticular vascularisation with spermatogenesis. The areas in testicular parenchyma with the greatest potential for spermatogenesis were found out using Power doppler Ultrasound which was using image based techniques. 3-D reconstruction was planned with the help of seven cross-sections of the PDUS images for constructing a spatial map of selective regions where the spermatozoa could be more available. This was possible only because the different zones of spermatogenesis corresponded with the vascular supply of the testis.[19]

Foresta[20] et al noted that the other vascular index namely PI of trans-testicular artery has increased resistances in men with azoospermia than with oligospermia. This was proved by the presence of normal testicular vessel distribution in obstructive azoospermia and the reduced or absence of normal vessels in unobstructed azoospermia. It is further extended that the presence of vascularity in the marginal zones might point to the presence of potential spermatogenic areas. Further, Battaglia[21] et al suggested that pulsatility index was noted to be significantly elevated in males with obstructive azoospermia (OA) in comparison to those suffering from unobstructive azoospermia(UOA). A color Doppler

based semi-quantitative scoring system has been recommended to distinguish obstructive from unobstructive varieties.

DOPPLER SEMI QUANTITATIVE SCORE

Category	*Number of intratesticular vessels visible*
0	No
1	1-3
2	More than 3 vessels

Various studies quoted above concluded that spectral intratesticular echo Doppler traces from the artery may associate with the spermatogenetic zones and also help in differentiation of OA from UOA radiologically.

Biaigotti[22] et al evaluated the usefulness of the PSV, EDV and RI of testicular vessels in distinguishing the various etiologies of altered sperm production and to find the association with follicle-stimulating hormone(FSH) and testicular volume if any.This study utilised the sperm production rate score(SPRS) which is calculated as:

$$SPRS = \frac{C \times V \times M/100 \times Mn/100}{D \times TV}$$

Where C= sperm concentration, V=volume of the ejaculate, M=Type A motility Mn=Morphologically normal, D=days of

abstinence and TV=Bilateral testis volume. The association between variables like RI, PSV, EDV, FSH level, each as independent variables in correlating as a function of SPRS was calculated. But the study had included varicoceles for causes of oligoasthenospermia and devised the various indices. RI and especially PSV, clearly identified obstructive and unobstructive azoospermia. Using angle dependent technique for measuring PSV or EDV, careful interpretation is needed for the assessment. But when RI is utilized to assess the vascularity within the testicular architecture, the above said limitation factor is eliminated.

The assessment of testicular Resistive Index can also be utilized as a diagnostic criteria for inflammatory diseases of the scrotum in humans.Jee[23] et al questioned the efficacy of the RI of the epididymal and intratesticular arteries as an analytic measure in the inflammatory disease of testis and its appendages. The RIs of the testicular artery in epididymitis associated with orchitis were considerably inferior to those in normal control subjects and in epididymitis alone patients ($p<0.01$). But, the RIs of the testicular artery in epididymitis alone and control subjects were alike. ($p>0.5$).This study proved that the diagnostic potential was highly precise in inflammatory diseases of the scrotum when the

Resistive index of both the vessels of the testis and the epididymis were between<0.5 and<0.7. Lefort[24] *et al.* studied the CDUS characters of testicular infarction caused by orchiepididymitis in selected patients over a 2-year phase, and remarked that an increased RI may indicate ischaemia.

Azospermia has been traditionally classified into obstructive azospermia and unobstructive azoospermia. Unobstructive azospermia (UOA) is represented by primary testicular failure. The structure of testicular parenchyma is significantly impaired in unobstructive azoospermia and few authors have showed that intratesticular blood flow is also characteristically altered and shows decreased or absent arterial flow, while people suffering from obstructive azospermia, generally have normal spermatogenesis and intact testicular blood flow.

By definition of OAT[25], there is no identified causative factor illuminating on the atypical quality of the spermatozoa. Hypothesis has been postulated that some patients with idiopathic oligozoospermia may have inexplicable partial obstruction to transport of sperm in the epididymis. Roughly 30% of the infertile men are affected by idiopathic oligoasthenoteratozoospermia (iOAT).

Semen volume along with sperm motility, gradually start declining around the age of twenty-two years upto eighty years, but not sperm concentration, with no evidence of a threshold. It is also noted that the levels of PSA,PAP, fructose and zinc are decreased in seminal concentration and the seminal activity of neutral alpha-glucosidase is on a declining trend in isolated asthenospermia. However, history of hernia repair and cryptorchidism, accessory gland infection mainly by Chlamydia trachomatis, genomic alterations in gamete, mitochondrial alterations also play a minor etiological role. Numerous patients with oligozoospermia linked with partial obstruction also display anti-sperm antibodies in serum, and they are identified to have infertility due to immunological reasons. Certain cases reveal aberrations in the region of rete testis that can be verified by vigilant screening by echography. Generally, the structures of the rete appear enlarged, mostly due to aberrations in embryological development. Administering oestrogen-like hormones during pregnancy can result in this condition, as shown in animal experiments. Also, aberrations in embryological maturation in the regions of the rete and efferent ductules of the testicular architecture, can result in oligospermia, cryptospermia or azoospermia in humans due to prenatal defects caused by the

consumption and accretion of hormone disrupting factors in the the antenatal period.

Hence, spermatogenesis is determined by various factors, from the hormonal milieu to the intratesticular blood flow. So, resistive index is a factor which combines all the important regulating factors such that its measurement takes an important role in assessing the spermatogenesis.

MATERIALS AND METHODS

TITLE OF THE STUDY

Assessment of the intratesticular resistive index by colour doppler ultrasonography measurment as a predictor of spermatogenesis

PERIOD OF STUDY

March 2012 to February 2013

STUDY DESIGN

Prospective diagnostic study

PLACE OF STUDY

The study was conducted in the Department of Urology and in Barnard Institute of Radiology, Madras Medical College and Rajiv Gandhi Government Hospital, Chennai- 3

ETHICAL CLEARANCE

The institutional ethical review board at our hospital approved the study.

No: 22032012.

The type of the present study is prospective diagnostic study. In all, 100 men (aged 22 -40 years, overall 200 testicles), 50 patients, who have abnormal sperm count in the form of mild oligoaesthenospermia according to WHO guidelines, 2009 (oligoasthenozoospermia defined as sperm concentration <15 $x10^6$ ml and reduced sperm motility in the form of percentage of progressively motile spermatozoa being < 32% or the percentage of progressively motile added with the non-progressive motile spermatozoa being < 40%).The other 50 men have normal sperm count , having attained paternity recently within 2- 15 months before recruitment were taken as controls.

All patients were clinically assessed by medical and surgical history(especially history of undescended testes, genital infectious disease, trauma, operations),lifestyle habits (smoking ,alcohol, etc),physical examination, three semen analyses (out of which the best values in sperm concentration and sperm motility is taken for evaluation),scrotal gray-scale USG and CDUS investigations. The patients were also sent for the endocrine evaluation to evaluate Serum testosterone (total), FSH, LH and Prolactin. Testosterone is measured by radioimmunoassay and the other hormones were evaluated by chemiluminescence method.

Scrotal US and CDUS were undertaken in a warm room with the patient supine, the penis resting on the lower abdomen. Each testicle was measured in three dimensions and the volume calculated. Doppler flow was measured in each testis using a trans-scrotal approach with a 10-MHz linear array probe (Esaote my Lab 60,Genova-Italy) (fig-8).

The PSV and EDV were calculated by the machine, recorded bilaterally for each patient, and expressed in cm/s and using the angle correction along the course of the artery. The investigated intratesticular artery had to be visualized for ≥ 0.5 cm. RI was then calculated as PSV-EDV/PSV. Atleast three RI values were recorded at the superior, middle and inferior poles of each testis. Minimally four doppler wave forms were recorded from each centripetal artery (fig-9).

It was also given importance that the study was conducted in the morning from 08:00 hours till 11:00 hours to avoid any effects of circadian rhythmicity on testicular blood flow.

The study was conducted at the Bernard Institute of Radiology, Rajiv Gandhi Government General Hospital & Madras Medical College, Chennai.

Fig-8: Measurement of RI using ESAOTE CDUS machine

INCLUSION CRITERIA

Patients were included if their major complaint was primary infertility, due to mild oligoasthenozoospermia (according to WHO guidelines, 2009), after 12–45 months of unprotected sexual intercourse.

The controls are 50 men who are in age-matched groups as the cases and who had normal sperm count and who had attained paternity 2-14 months before recruitment.

EXCLUSION CRITERIA

1) History of testicular trauma.

2) Surgery on the scrotum or testes like hydrocoele surgery or varicocelectomy.

3) Men clinically suspected of having obstructive infertility due to a vas deferens obstruction by palpation or TRUS.

METHOD OF STUDY

Institutional Ethics Committee approval was obtained. Informed consent was taken from all patients. All details were recorded as per the proforma (Appendix-3). All the patients and controls were taken up for scrotal US and colour doppler ultrasonogram and both the testes were measured their volume and then using the 10MHz linear probe, doppler spectral traces were obtained in both the testes in the three poles and the PSV and EDV were measured and RI was calculated using the formula: RI= PSV-EDV/PSV. All the readings were recorded in the respective proforma for both groups. Hormonal analysis results were also noted.

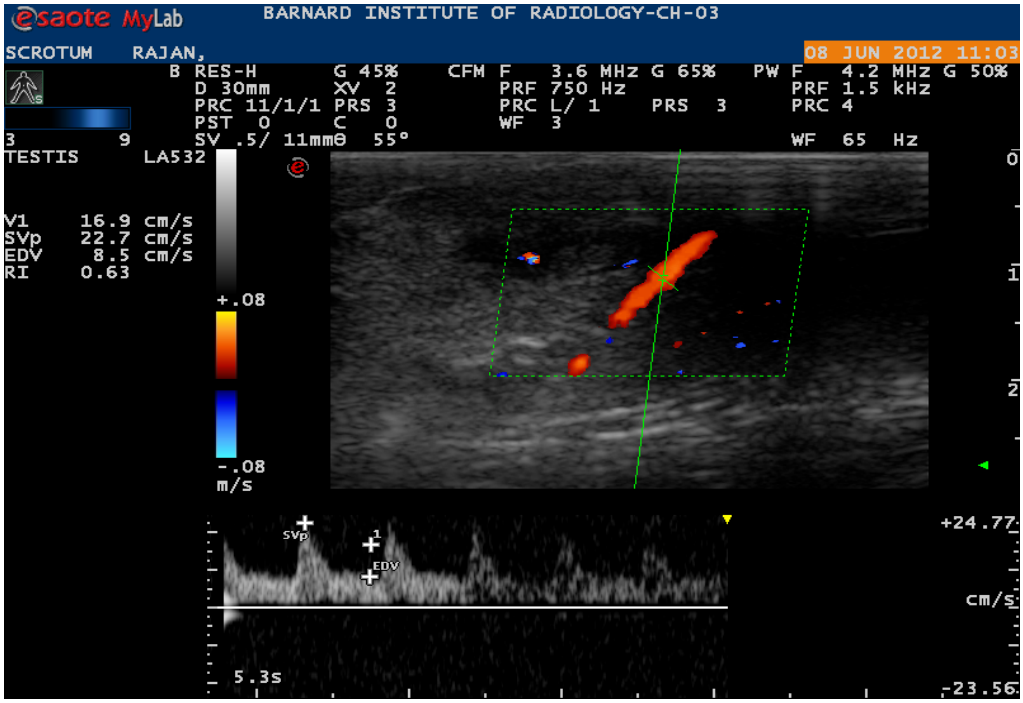


Fig :9- RI calculation by the CDUS machine ESOATE

STATISTICAL ANALYSIS

After the study, correlation co-efficient of RI is compared with other parameters like semen analysis, hormonal assay, etc and Independent-T test is applied for expressed values and to estimate P value and P value of ≤ 0.05 is considered as statistically significant.

RESULTS

The age of the study group ranged between 22 and 40. Among the oligoasthenozoospermia cases and normal controls, majority 38 out of 50 were between 26 and 35 of age. The least number among both the peers was between 36 to 40.(5 cases)-[table 1 and fig:10]

Table-1: showing the age matched distribution of cases and controls.

		Case	Control	Total
Age Groups	21-25	7	7	14
	26-30	22	22	44
	31-35	16	16	32
	36-40	5	5	10
Total		50	50	100

Fig-10: Age distribution of Oligoasthenozoospermia cases and Normal Sperm Count controls

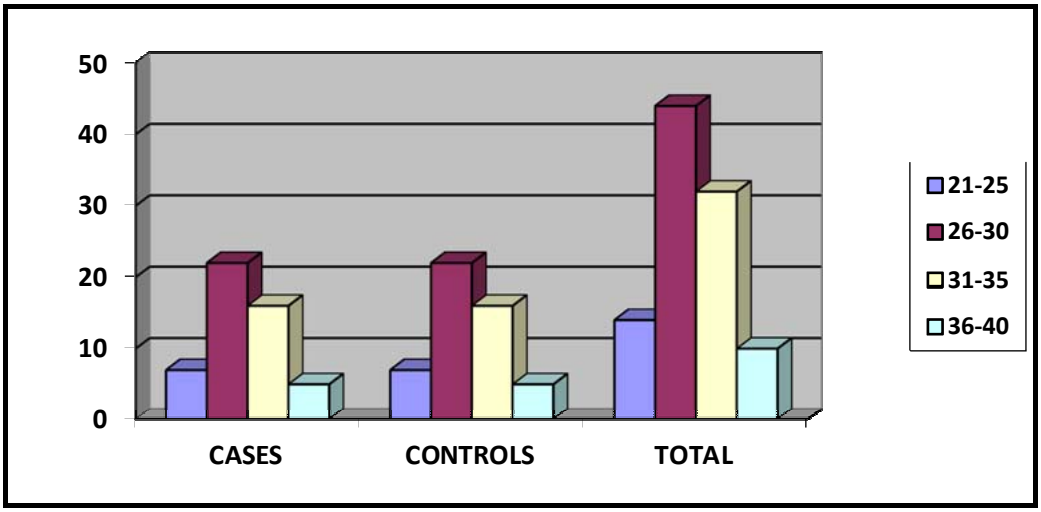


Table-2: Mean and SD values of the Semen Analysis of cases and controls

Semen parameters	Cases N=50		Controls N=50	
	Mean	**SD**	**Mean**	**SD**
Semen Volume(ml)	2.75	0.456	3.32	0.543
Semen Concentration(Million/ml)	13.32	1.467	60.77	14.03
Semen Motility(Type A)	19.60	6.14	51.30	12.19

The mean semen volume for the cases of oligoasthenospermia cases was 2.75 ml with a standard deviation of 0.456 whereas the controls had 3.32 ml as the mean semen volume with a SD of 0.543. (fig 11).The sperm concentration in the cases was found to have a mean value of 13.32 million/mL with a SD of 1.467 where as the controls had 60.77 million/mL as the mean sperm concentration with a SD of 14.03.(Table-2 and Fig-12)

The sperm motility of progressiveness, i.e Type A motility which defines the group of OAS as < 32% according to WHO guidelines,2009 was found to be 19.6% with a SD of 6.14 in cases and 51.3% with SD of 12.19 in controls.(Fig:13)

Thus the cases and control groups were stratified and then they were subjected to USG and CDUS scrotum and the respective

parameters of testicular volume followed by the measurement of PSV and EDV in the upper, middle and lower poles of both testes and recorded. Since the CDUS machine has an inbuilt software to calculate RI, all the values were also recorded and the mean and standard deviation was arrived at.

SEMEN VOLUME (ML)

Fig11: showing the semen volume among cases and controls

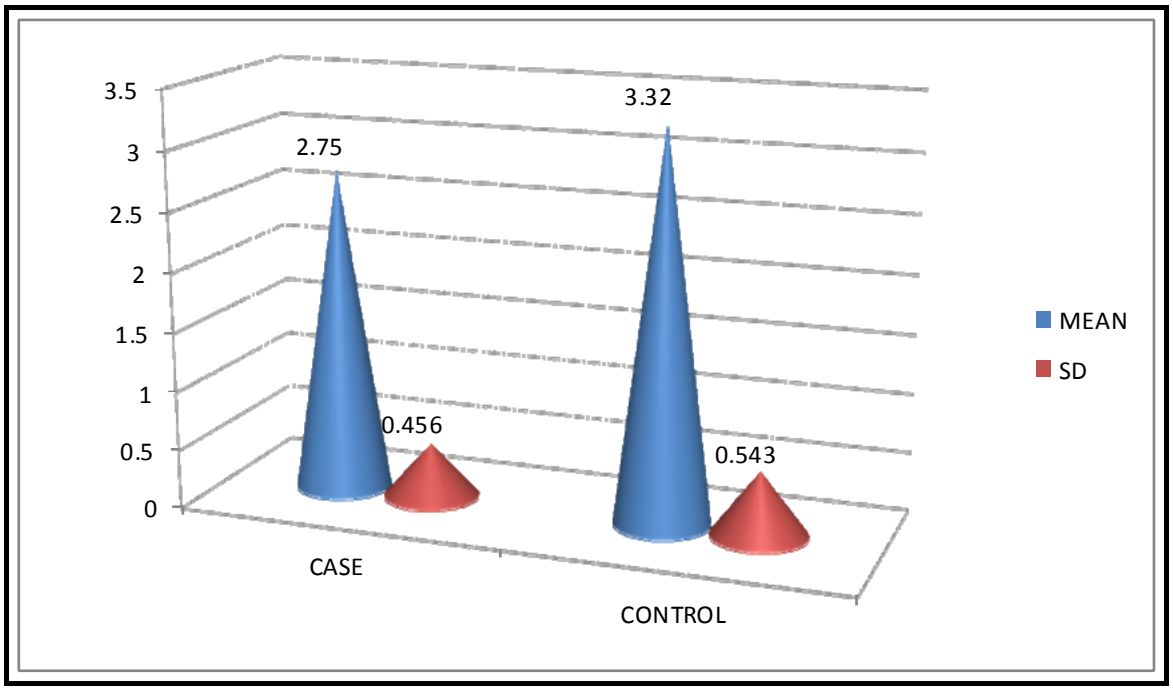


Fig 12: Sperm Concentration (millions/mL) among cases and controls

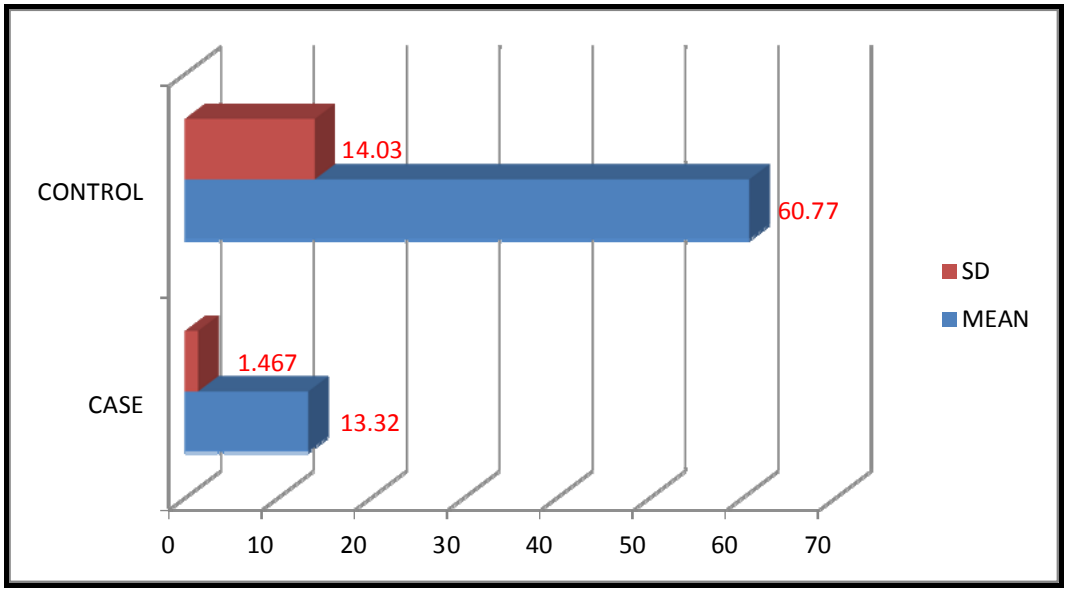
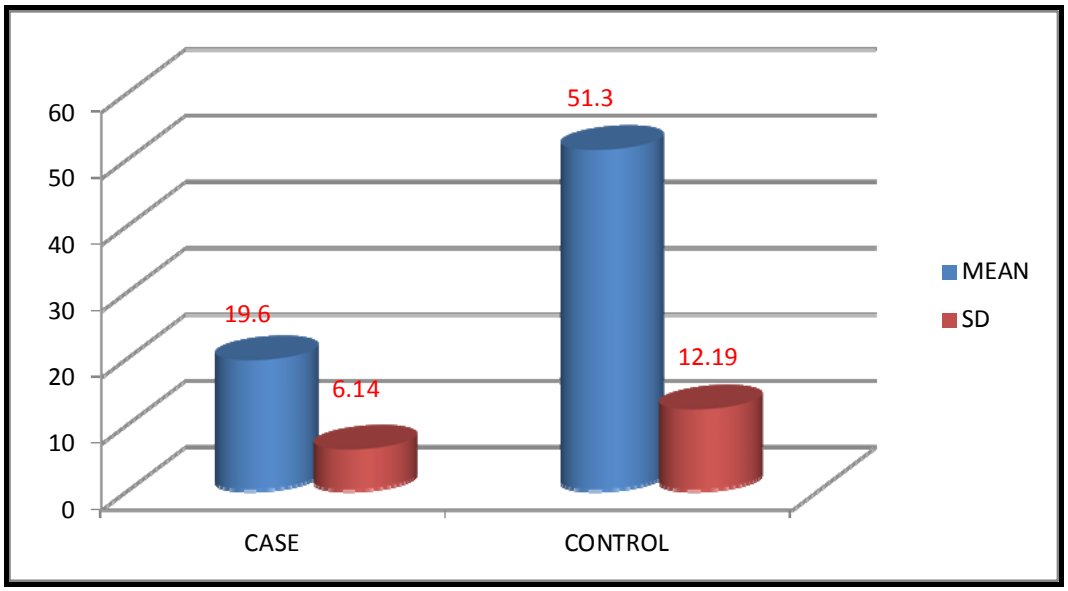


Fig 13: Sperm Motility (Type A-Progessively motile%) among cases and controls



The testicular volume was measured by USG scrotum on both the testes and the mean values were taken. The mean value of the testicular volume in cases is 18.24 ml with a SD of 0.391.

The mean testicular volume in controls was 18.39 ml with a SD of 0.397. When test of significance was applied on this factor, it was found to have a P value of >0.05. Thus, the testicular volume did not have any significant association with spermatogenesis. (Fig 14 & 15).

Fig 14: Testicular volume among cases and controls

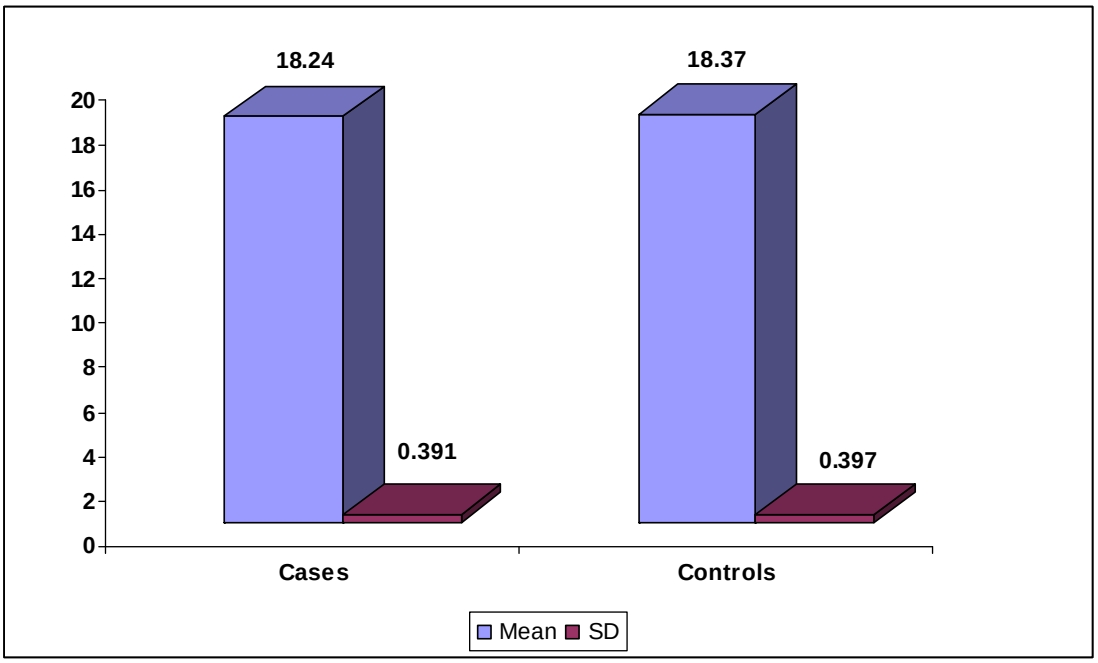


Fig 15: Testicular volume among cases and controls

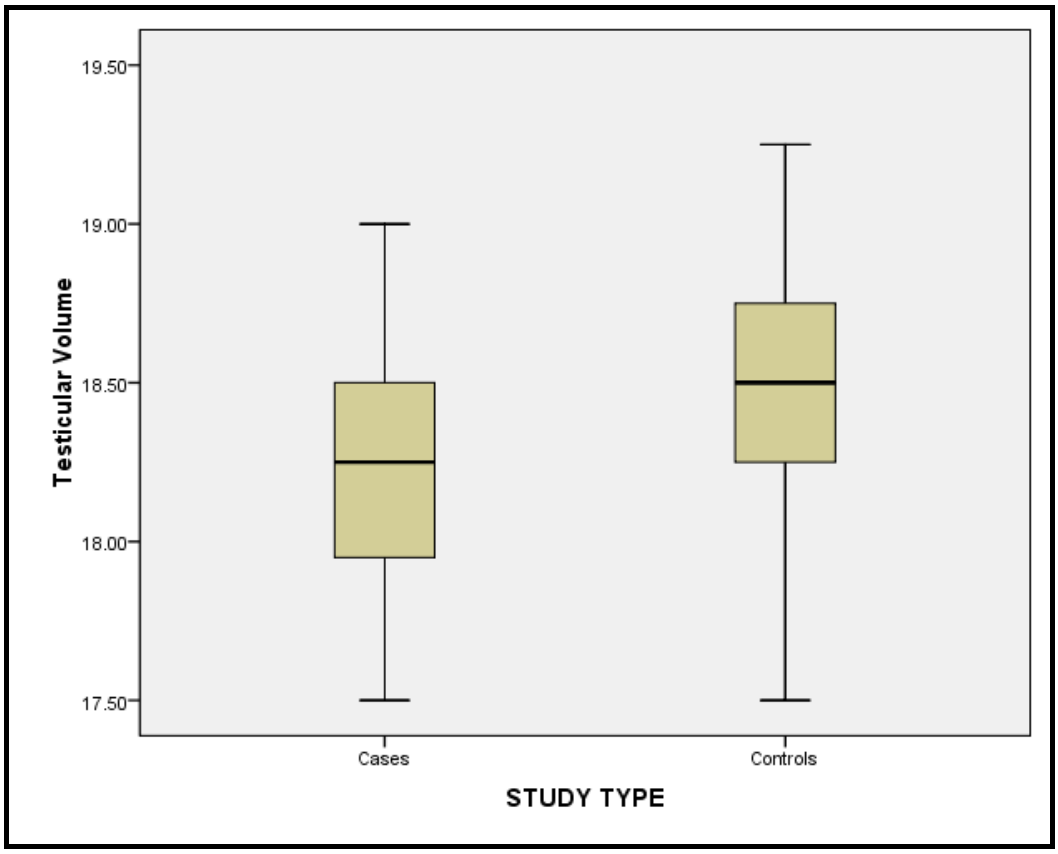


Table 3- showing distribution of hormone values among the study groups

	Cases N=50		Controls N=50	
	Mean	**SD**	**Mean**	**SD**
Testosterone(Total)	5.15	1.215	5.22	1.82
FSH	7.67	2.42	6.31	3.64
LH	6.08	1.43	5.57	2.46
Prolactin	7.96	3.01	7.21	2.70

Hormonal analysis was also done in both cases and controls. (Tab:3) Serum testosterone values (total) were compared between cases and controls. The cases had a mean value of 5.15 units with a SD value of 1.215 where as the controls had a mean value of 5.22 with a SD of 1.82. (Fig:16)They were not found to have significant association. (Fig:17)

Fig:16- Testosterone values among cases and controls

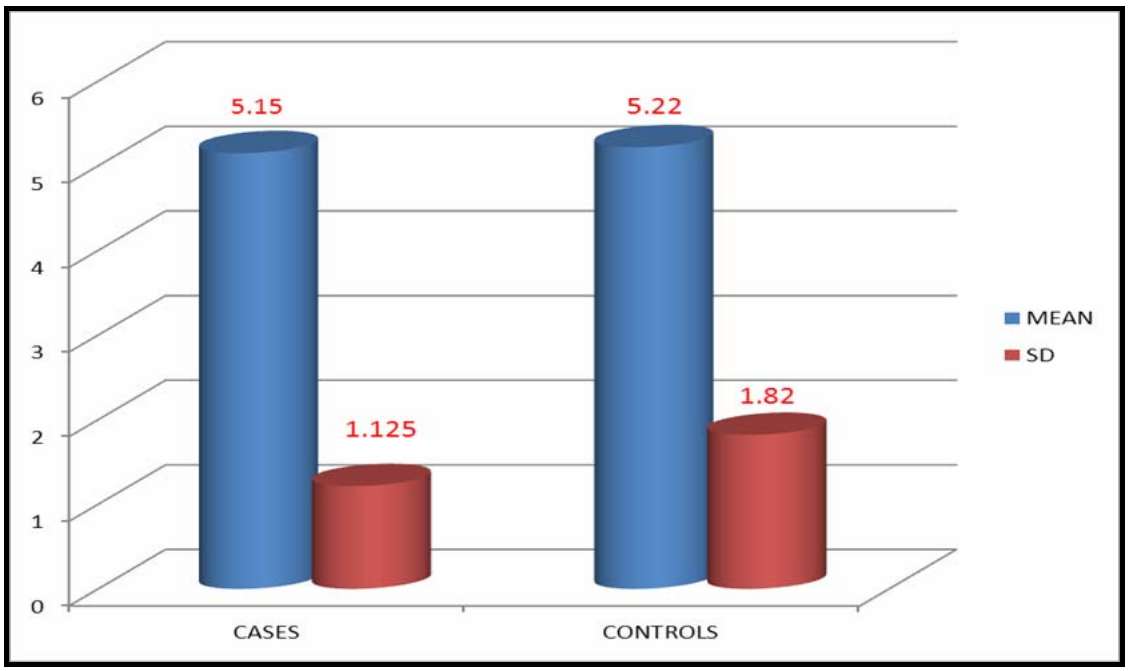


Fig:17- Testosterone values among cases and controls

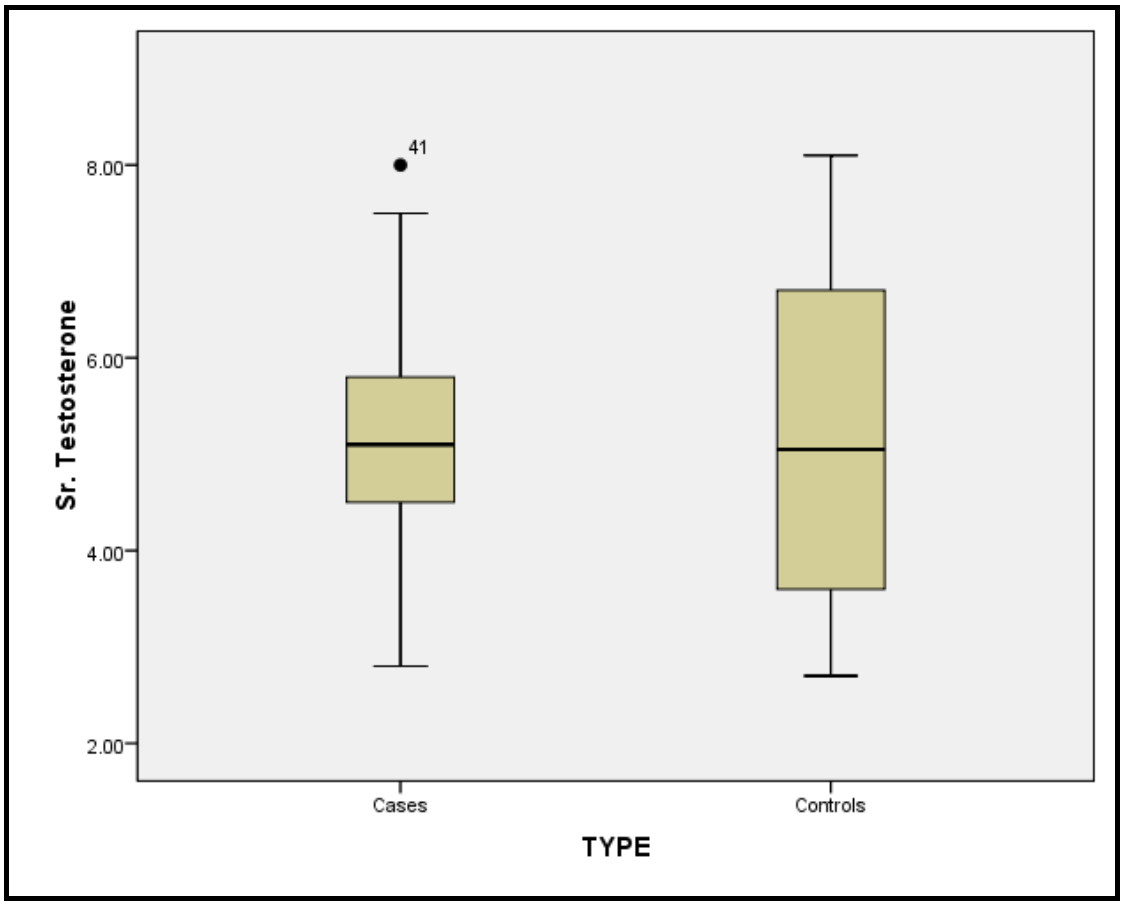


Fig:18 - FSH values among cases and controls

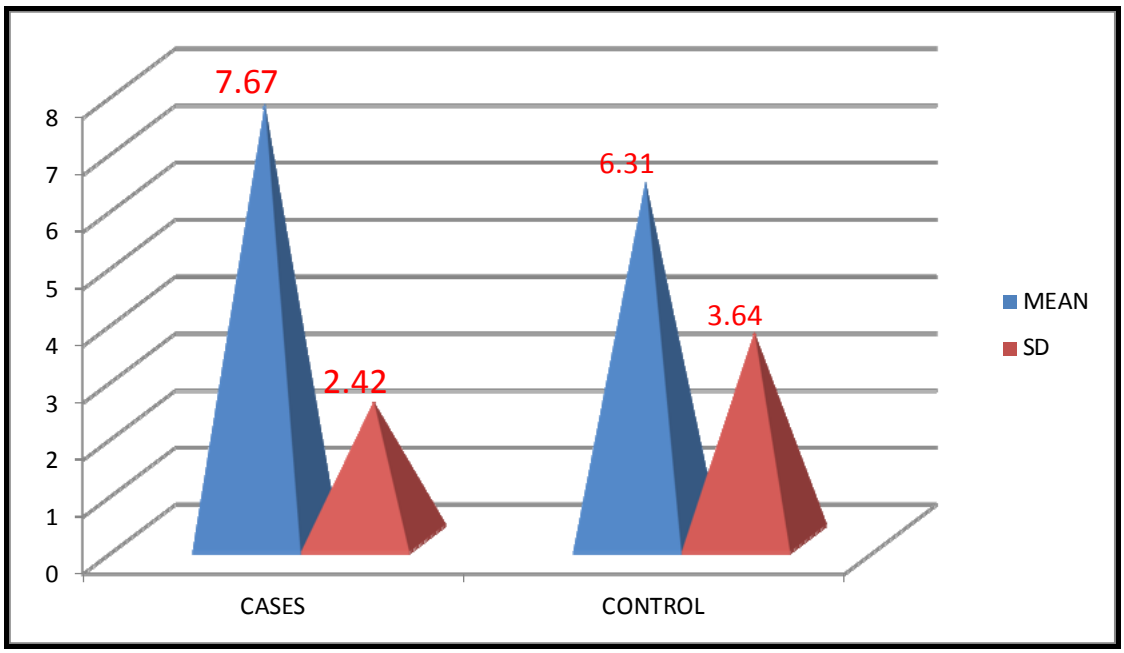
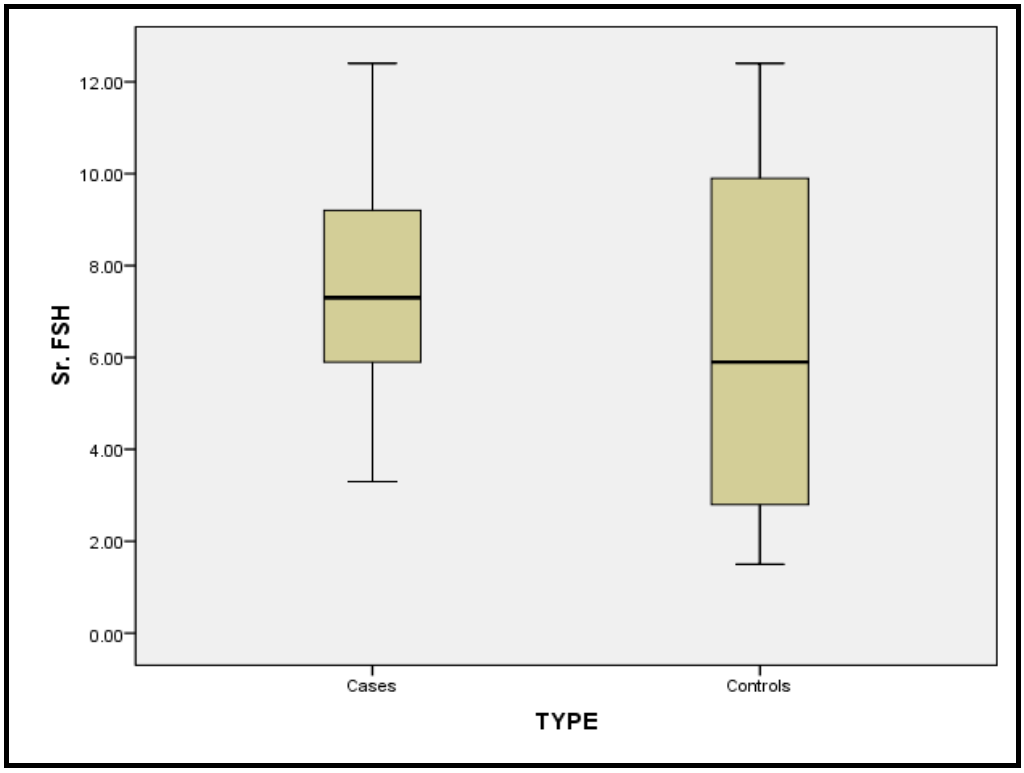


Fig:19- FSH values among cases and controls



Similarly other hormones like FSH, LH and Prolactin were also compared.(Tab :4) All the mean values with the SD values are tabulated and graphically represented above. The mean value of FSH in cases and controls (Fig-18 & 19) is 7.67 and 6.31 IU respectively and the SD values are 2.42 and 3.64 respectively and the difference between the cases and controls is found to be significant (P value:0.038). LH and prolactin values are tabulated and their mean and SD values are noted respectively in the bar diagram. (Fig 20 &21).

Fig :20- LH values among cases and controls

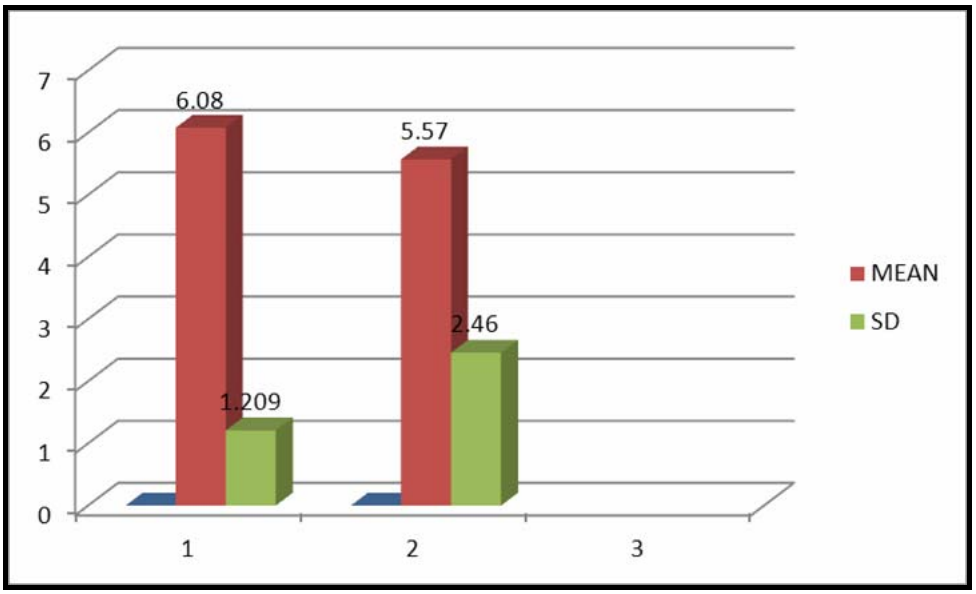
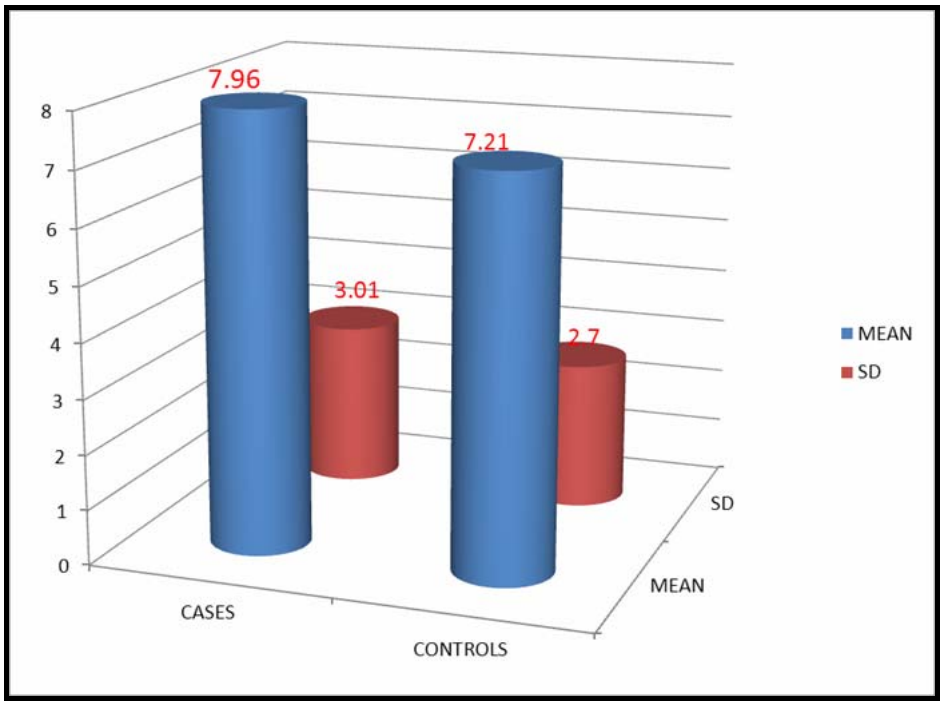


Fig:21- Prolactin values among cases and controls

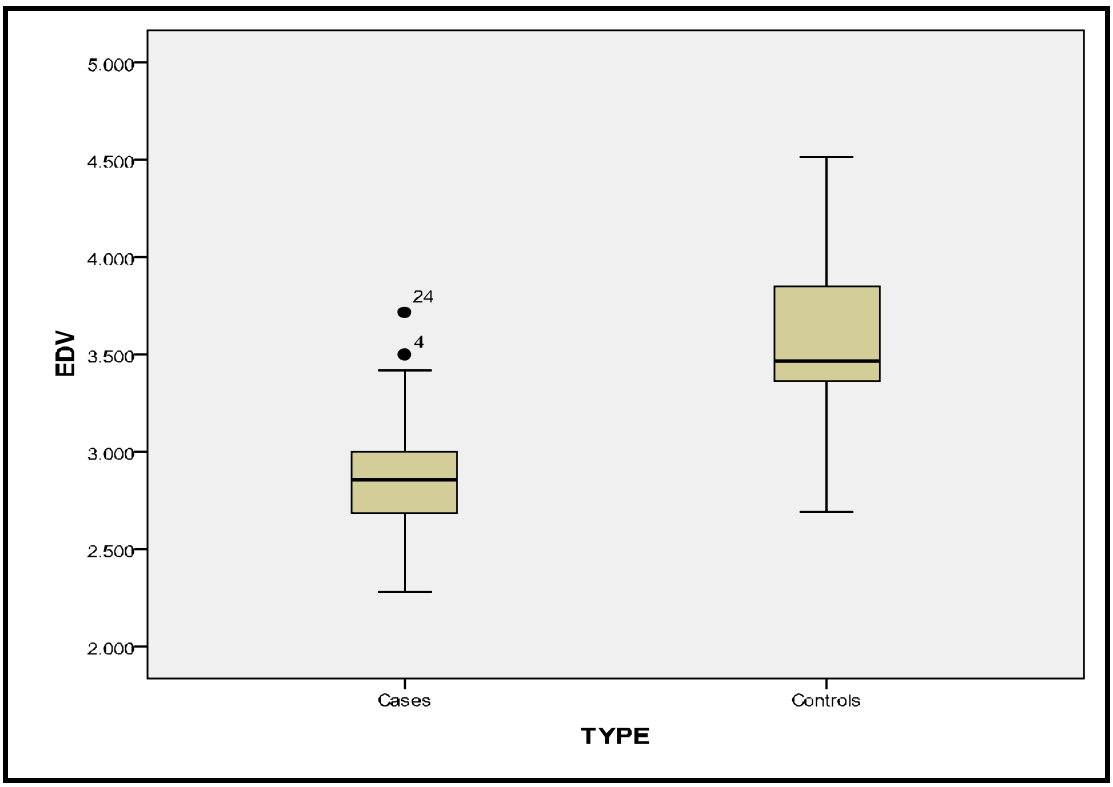


Also testing the significance by T-test of the various hormones as tabulated below, the levels of FSH was found to be fairly associated (P value 0.038) but LH and prolactin showing no association(P value >0.05) among the cases and controls.

Table-4: Group Statistics of Hormonal Values

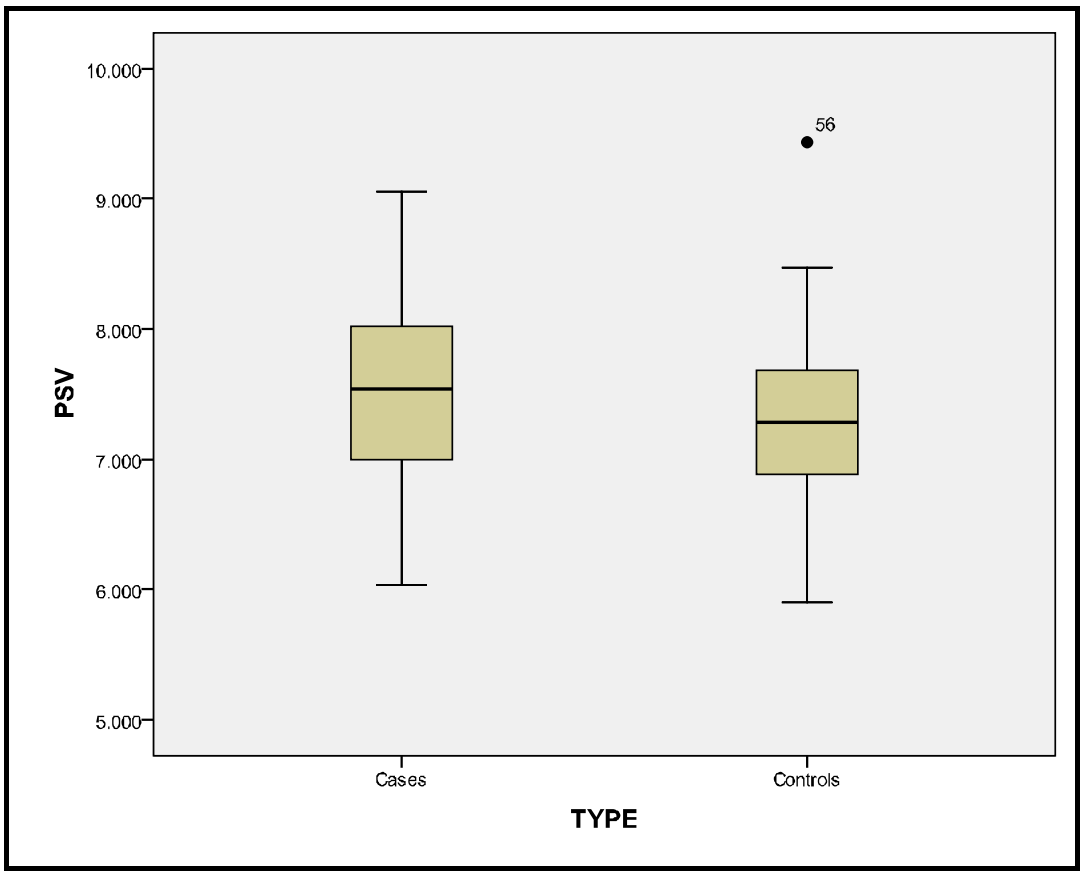
	TYPE	N	Mean	Std. Deviation	Std. Error Mean
Sr. Testosterone (Total)	Cases	50	5.1518	1.21543	.17189
	Controls	50	5.2220	1.82950	.25873
Sr. FSH	Cases	50	7.6660	2.42644	.34315
	Controls	50	6.3120	3.64885	.51603
Sr LH	Cases	50	6.08	1.2091	.20293
	Controls	50	5.5680	2.46025	.34793
Prolactin	Cases	50	7.9578	3.01540	.42644
	Controls	50	7.2116	2.70671	.38279

Fig 22- showing the box-plot of end diastolic velocity(EDV) among cases and controls.



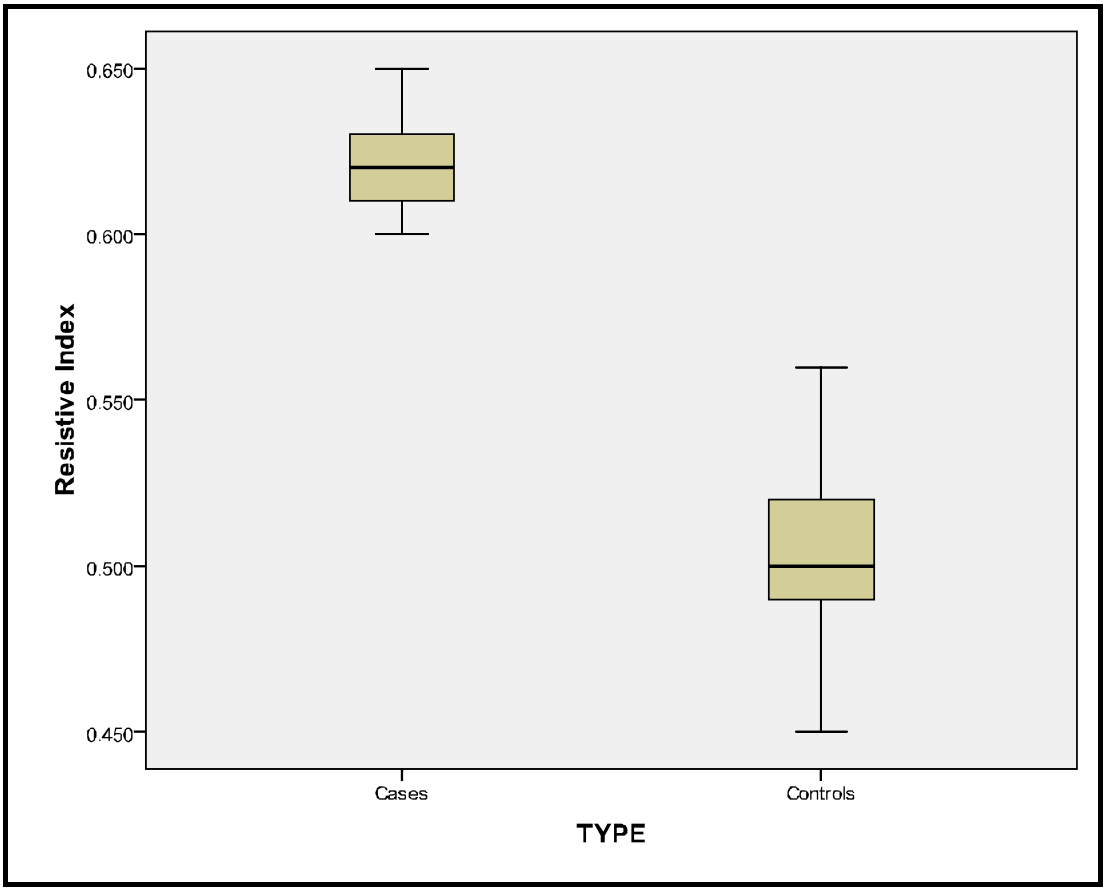
On measuring and tabulating mean EDV (Fig-22) among cases and controls , the mean value is found to be 2.86 with a SD of 0.285 in the former and 3.53 and 0.363 respectively in the later. Though the association was found to be slightly significant, the meaurment of RI carries more significance than EDV.

Fig23- Peak systolic velocity (PSV) among cases and controls



Similarly when PSV(Fig:23) is used for calculating RI,the mean values of the cases is found to be 7.51 with a standard deviation of 0.662 in cases and 7.31 and 0.693 respectively in controls.The association was not statistically significant here.

Fig :24- Resistive Index among the cases and controls



Finally arriving at the calculation of RI(Fig:24) among cases and controls using the variables of PSV and EDV ,the mean value in the cases is found to be 0.62 with a standard error of 0.001. The median value is also found to be 0.62.The standard deviation is found to be 0.0126. The minimum value among the cases is found to be 0.6 and the maximum is found to be 0.65.

When the values were calculated for the controls, the mean is found to be 0.507 with a standard error of 0.003.The median value is 0.5 and the standard deviation is 0.0245.The minimum value

among controls is 0.45 and the maximum value is 0.56.Applying the test of significance, the P value is found to be < 0.05 and it is finally proved that the resistive index has a strong association with spermatogenesis.

DISCUSSION

The powerhouse of the sexual function and the producer of the male sex gametes, the testes receive their blood supply from the paired gonadal arteries namely the internal spermatic arteries. Both the testicular arteries pass obliquely over the ureter after arising from the abdominal aorta, and the distal part of the external iliac vessels to enter the internal inguinal ring, and passes through it, along with the other contents of the spermatic cord.

After giving rise to two or three of the branches of testicular artery, which travel along the ductus deferens, and supply the epididymis, and later undergo anastamoses with the artery to the vas deferens; others branches penetrate the posterior aspect of the tunica albuginea, and supply the parenchyma of the testis. The testis needs adequate vascular supply for the functions of spermatogenesis and steroidogenesis and its maturation per se.

In the present study, it was found beyond doubt that the occurrence of oligoasthenospermia as the reduced sperm concentration and motility, owes to the decreased blood supply to the gonads which is proved by the increased resistive index. The underlying pathology must be ischaemia leading to damage of the

organ subclinically. This finding is concordant with most of the parallel studies depicting the occurrence. The most prominent of them is the similar study by Germar-Michael[26] et al(Austrian study) who were able to demonstrate the above finding having 80 patients and 80 age-matched controls. Also, an indirect evidence was pointed to this finding when testicular artery restriction was done in bulls, leading to near total arrest of spermatogenesis and the alteration in histology.

Comparing the semen analysis between the present study and the Austrian study, the semen analysis in the present study revealed mean sperm concentration of the cases is 13.32 million/mL whereas in the Austrian study it was 13.2 million /mL which is comparable. The sperm motility in the former is found to be 19.6% Type A whereas the later had 12.3%.The higher percentage in the former is due to the inclusion of progressively motile sperms upto 32% according to WHO 2009 guidelines. The former was according to WHO 2002 guidelines. The testicular volume in the Austrian study was found to be 16.8 mL with standard deviation of 6 whereas in the present study it is 18 mL but with SD of <1.This variance might be due to the patient selection and in general, the Indian

males have lesser testes size since the maximum testicular volume inclusive of cases and controls is only around 20 cc.

The advent of CDUS has led to the convenience and rapidity in assessing the blood flow in any organ. In our study, the CDUS machine used was the most innovative ***Esaote my lab 60,*** which paralled with the machine used in the Austrian study (Sequoia 512, Acuson Siemens, Germany). The probe used in the Sequoia machine was 14 MHz where as in the present study the probes of 10 MHz were used for the study. Decreased microcirculation in the testes has been proved in clinical varicoceles by CDUS by demonstration of increase in RI. It goes beyond doubt that testicular torsion should result in increased RI due to ischaemia as experimented by few studies. Lefort et al also extended the theory on testicular ischaemia caused due to inflammatory conditions like epididymoorchitis where RI was found to be elevated due to testicular infarct[24].

The testicular RI was also used in some studies to evaluate the testicular integrity postoperatively. Tanirvedi[27] et al evaluated the testicular bloodflow with more concern for the microcirculation by evaluating RI in 56 patients who underwent high and low microsurgical varicocele repair. The low repair was better in maintaining the microcirculation even after surgery. Beddy[28] et al

proved the inguinal hernioraphy both by open and laparoscopic methods did not affect the reversal of testicular function and decrease in testicular resistance which was demonstrated by CDUS.

With so much of evidence to back the clinical utility of RI in assessment of testicular microcirculation by evaluatiuon of RI, it was slowly used as an non invasive tool to predict the vital function of sperm production since it depends on the microcirculation.

RI, being used as predictor of spermatogenesis, when evaluated in men with normal sperm counts, have shown the values as below (Table-5).

COMPARISON OF THE TESTICULAR RI-S IN VARIOUS STUDIES

Table-5: Comparison of RI-s in various studies in normal males

Study	Testicular RI
Jee et al	0.5
Atilla et al	≥0.5
Biagiotti et al	≥0.5
Germar et al	0.54
Hassan et al	0.56
Present study	0.55

All the values mentioned above stand for the adult males. The same index has been tested in young boys by Paltiel[29] et al. Maturational changes were evaluated using CDUS. It was noted that in the testes of <4 mL, the mean RI was 0.87 and on the contrary, in the testes of > 4 mL, the mean RI was 0.57.So,this norm of the blood flow which holds good for adults may not be applicable for prepubertal boys especially in testes < 4 mL. Hence the results of this study would only apply to males after puberty, which is similar to the Austrian study.

In my present study, the mean testicular RI in oligoasthenospermic males has been found to be 0.62. (>0.6 with P value of <0.001 which is statistically significant).In the parallel studies by Germar[26] et al and Hassan[30] et al ,the mean RI was found to be 0.68 and 0.67 respectively with statistical significance. The RI value in our South Indian study is found to be lesser. This may be accounted for the difference in the testicular arterial luminal diameter between Indian males and the western males. But the testicular volume in the present study between cases and controls is nearly same, around 18 cc. The western studies also prove the same that the testicular volume is not related to spermatogenesis. The pioneer studies of Atilla[2] et al and Biagotti[3] et al also went on to

prove that there was no volume differences between the undescended testes and males with OAS respectively.

The former measured arterial impedance and testicular diameter with the use of color Doppler ultrasonography on 22 adults with undescended testes keeping arterial impedance of the contralateral descended testes as controls. Later inguinal orchiectomy was perfomed in all patients and histological examination of the testes were done. He concluded that the arterial impedance of an undescended testis as measured by the RI of the testicular artery using CDUS provides better information on the histology than the volume.

The later assessed whether the peak systolic velocity (PSV), end diastolic velocity (EDV) and resistive index (RI) of testicular arteries may be useful in distinguishing the various causes of dyspermia when compared with follicle-stimulating hormone(FSH) and testicular volume. These two studies proved beyond doubt about the importance of Resistive index as the predictor of spermatogenesis.

The semen analysis in the present study revealed mean sperm concentration of the cases is 13.32 million/mL whereas in the Austrian study it was 13.2 million /mL which is comparable. The sperm motility in the former is found to be 19.6% Type A whereas the later had 12.3%.The higher percentage in the former is due to the inclusion of progressively motile sperms upto 32% according to WHO 2009 guidelines. The former was according to WHO 2002 guidelines. The testicular volume in the Austrian study was found to be 16.8 mL with standard deviation of 6 whereas in the present study it is 18 mL but with SD of <1.This variance might be due to the patient selection and in general, the Indian males have lesser testes size since the maximum testicular volume inclusive of cases and controls is only around 20 cc.

EFFECT OF HORMONES

The role of FSH is to induce permanent maturational effects in the sertoli cell and the seminiferous epithelilum as in normal puberty. The synergistic action of LH/testosterone and FSH is necessary for the initiation, maintenance and also for reinitiation of normal spermatogenesis. FSH either above >10 IU in severe oligospermia or azoospermia may indicate testicular dysfunction or non-obstructive azoospermia. If <1.5 IU, it may indicate

hypogonadotrophic hypogonadism. In the present study, the levels of LH parallel the cases and controls, which go to say they have no association. This finding was similar in the parallel study relating the insignificance. Similarly, the levels of total testosterone and prolactin behave the same way in the present study and the parallel studies indicating their very poor association. The difference in the levels of FSH between the cases and controls found to be significant (P value:0.038) as there is possibly mild stimulation of the gonadotrophin in OAS individuals and hence a slightly raised FSH in pathological spermiogram. Hence overall, hormonal analysis may not have significant association with spermatogenesis.

Having ruled out any obvious predictors measured in the present study in influencing the outcomes in the oligoasthenospermic individuals. Resistive index seems to be the sole phenomenon which is a ready reckoner for its noninvasive and easy technique of evaluation of infertility and it has definitive value in the follow up and looking at the prognosis after treatment.

In the present study, due to limited resources, karyotyping could not be done, hence a minimal bias due to genetic overlap could not be overcome. This is the only limiting factor in the study.

But the pathogical spermiogram patients in the Austrian study were evaluated for karyotyping using cultures of peripheral lymphocytes.Though the major Y microdeletions, i.e., severe oligoaesthenospermia and azoospermia individuals were already excluded ,the karyotyping by chromosomal analysis could identify four men out of eighty (<3%) to possess abnormal karyotypes.

CONCLUSION

1) Intratesticular resistive index measurement by Colour Doppler Ultrasonogram in oligoasthenospermic patients has revealed a value of >0.6 which is comparable with similar parallel studies conducted worldwide. These results suggest that CDUS RI parameter can be used as a simple, safe, rapid and non invasive predictor test for spermatogenesis and infertility.

2) Other parameters like testicular volume, serum testosterone (total), FSH, LH or Prolactin have also been studied and they were not significantly associated with the outcomes except FSH. The difference in the levels of FSH between the cases and controls is found to be significant which means that the OAS patients may have a slightly raised FSH compared to males with normal spermiogram.

3) Hence Resistive index can be considered to be a possible diagnostic criterion to assess the infertile males and an useful tool for follow up.

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Master Chart (Cases)

Master Chart (Control)

Clr W.P.No: 6,8-9,15,21,33,35-36,38-48

B&W W.P.No: 1-5,7,10-14,16-20,22-32,34,37,49-59

B&W W.O.P.No: 60-81

CONSENT FORM

Study Title

ASSESSMENT OF THE INTRATESTICULAR RESISTIVE INDEX BY COLOUR DOPPLER ULTRASONOGRAPHY MEASURMENT AS A PREDICTOR OF SPERMATOGENESIS.

I, _____ hereby give consent to participate in the study conducted by Dr.S.AHMED MARZOOK, 2nd Year M.Ch (Urology) Postgraduate Student, Madras Medical College, and Rajiv Gandhi Government General Hospital, Chennai-3 and to use my personal clinical data and result of investigation for the purpose of analysis. I also give consent for further investigations.

Signature / Thumb Impression of the patient/ relative Place Date

Patient Name and Address

Signature of the Investigation

PROFORMA

NAME OF THE PATIENT: AGE : OP NO:

Occupation: H/o smoking / Alcohol: Diabetic/Hypertensive

SEMEN ANALYSIS:

Macroscopic Examination:

Colour:

pH:

Volume:

Viscosity:

Liquefaction:

Microscopic Examination

Actively Motile:

Sluggishly Motile:

Non-Motile:

Movement:

Sperm Count:

Normal morphology:

Abnormalities:

TESTICULAR VOLUME (USG): Right: Left:

COLOUR DOPPLER USG MEASUREMENT

RT TESTIS	PSV	EDV	RI	AVERAGE
UPPER:				
MIDDLE:				
LOWER:				
LT.TESTIS				
UPPER:				
MIDDLE:				
LOWER:				

HORMONAL ANALYSIS

SR. TESTOSTERONE:

FSH:

LH:

PROLACTIN:

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI -3

Telephone No : 044 25305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr. S. Ahmed Marzook
PG in MCH Urology
Madras Medical College, Chennai -3

Dear Dr. S. Ahmed Marzook

The Institutional Ethics committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled " Assessment of the intratesticular resistive index by colour Doppler ultrasonography measurement as a predictor of spermatogenesis " No.22032012.

The following members of Ethics Committee were present in the meeting held on 22.03.2012 conducted at Madras Medical College, Chennai -3.

1.	Prof. S.K. Rajan. MD	-- Chairperson
2	Prof. Pregna B. Dolia MD Vice Principal, Madras Medical College, Chennai -3 (Director , Institute of Biochemistry, MMC, Ch-3)	-- Member Secretary
3.	Prof. B. Kalaiselvi. MD Prof of Pharmacology ,MMC, Ch-3	-- Member
4.	Prof. C. Rajendiran, MD Director , Inst. Of Internal Medicine, MMC, Ch-3	-- Member
5.	Thiru. S. Govindsamy. BA BL	-- Lawyer
6.	Tmt. Arnold Soulina MA MSW	-- Social Scientist

We approve the proposal to be conducted in its presented form.

Sd/ Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, and SAE occurring in the course of the study, any changes in the protocol and patients information / informed consent and asks to be provided a copy of the final report.

Member Secretary, Ethics Committee

ஆராய்ச்சி ஒப்புதல் கடிதம்

நிற டாப்ளர் மீயொலி வரைவின் (கலர் டாப்ளர் அல்ட்ரா சோநோகிராம்) மூலம் உள் விரையின் தமணி தடை குறியீடு (ரெசிச்டிவ் இன்டெக்ஸ்) மதிப்பீட்டினால் விந்தாக்கம் பற்றி முன்கூற்றி அறிதல் பற்றிய ஆராய்ச்சி

பெயர் : தேதி :

வயது : உள்நோயாளி எண் :

பால் : ஆராய்ச்சி சேர்க்கை எண் :

இந்த ஆராய்ச்சியின் விவரங்களும் அதன் நோக்கம் முழுமையாக எனக்கு தெளிவாக விளக்கப்பட்டது.

எனக்கு விளக்கப்பட்ட விஷயங்களை நான் புரிந்து கொண்டு எனது சம்மதத்தை தெரிவிக்கிறேன்.

நிற டாப்ளர் மீயொலி வரைவின் (கலர் டாப்ளர் அல்ட்ரா சோநோகிராம்) மூலம் உள் விரையின் தமணி தடை குறியீடு (ரெசிச்டிவ் இன்டெக்ஸ்) மதிப்பீட்டினால் விந்தாக்கம் பற்றி முன்கூற்றி அறிதல் பற்றிய ஆராய்ச்சிக்கு தேவையான அணைத்து விவரங்களையும் தெரியபடுத்துவதற்கு முழு சம்மதம் தெரிவிக்கிறேன்.

இந்த ஆராய்ச்சியில் பிறரின் நிர்பந்தமின்றி என் சொந்த விருப்பத்தின் பேரில் தான் பங்குபெருகிறேன் மற்றும் இந்த ஆராய்ச்சியில் இருந்து எந்நேரமும் பின் வாங்கலாம் என்பதையும் அதனால் எந்த பாதிப்பும் ஏற்படாது என்பதையும் நான் புரிந்து கொண்டேன்.

நான் என்னுடைய சுய நினைவுடனும் மற்றும் முழு சுதந்திரத்துடனும் இந்த மருத்துவ ஆராய்ச்சியில் என்னை சேர்த்துக்கொள்ள சம்மதிக்கிறேன்.

இந்த ஆராய்ச்சியின் தகவல்களையும் முடிவுகளையும் அறிவியல் நோக்கத்திற்காக பயன்படுத்துவதற்கு நான் அனுமதிக்கிறேன். நான் இந்த ஆராய்ச்சியில் பங்கு பெற சம்மதிக்கிறேன்.

பங்கேற்பவர் பெயர் :

பங்கேற்பவர் கையொப்பம்

(அ) இடது கட்டைவிரல் ரேகை

ஆய்வாளர் பெயர் :

ஆய்வாளர் கையொப்பம்

இடம் :

தேதி :

S NO	Name	Age	VOLUME	SPERM	MOT A	MOT A+B	BOTH	MORPHO	RU PSV	RU EDV	RU RI	RM PSV	RM EDV	RM RI	RL PSV	RL EDV
1	Shanmugam	27	4	15	+	-	-	25	8.1	2.9	0.63	8.5	3.4	0.6	9.7	3.7
2	Rajkumar	22	3	18	+	+	+	22	9.5	3.4	0.64	6.9	2.7	0.61	7.8	3.2
3	Perumal	32	3	13	+	+	+	23	7.7	3	0.61	7.9	2.8	0.65	9.2	3.5
4	Inbanathan	37	2.5	12	+	-	-	24	8.5	3.4	0.6	7.3	2.8	0.61	9.7	3.8
5	Ananthakrishnan	28	2.5	15	+	+	+	25	8.2	2.7	0.66	8.4	3.5	0.58	7.2	2.8
6	Arulmurugan	29	2	18	+	-	-	22	4.9	1.9	0.61	8.5	2.8	0.66	5.9	2
7	Arun prasad	24	3	18.5	+	-	-	25	6.3	2.1	0.66	5.4	2.1	0.61	6.5	2.6
8	Arunagiri	33	3	12	+	+	+	23	7.4	2.3	0.68	9.1	3.5	0.61	7.3	2.7
9	Santhosham	26	2.6	10.2	+	+	+	25	8.3	3.4	0.59	10.1	3.7	0.63	8.7	3.7
10	George mathew	33	2	13.9	+	+	+	23	5.4	1.7	0.68	8.3	3.7	0.55	6.3	2.3
11	Mathesh	30	3	14.6	+	+	+	24	9.8	3.9	0.6	7.5	2.8	0.62	6.8	2.5
12	Paraman	29	2.5	15.5	+	+	+	26	5.5	2.3	0.59	5.9	2.3	0.61	6.3	2.2
13	Aakash	22	3	15	+	+	+	23	9.1	2.9	0.68	6.2	2.2	0.64	8.7	2.9
14	Seetharaman	34	3.2	19	-	+	-	24	9.1	3.7	0.59	9.4	3.9	0.58	9.5	3.8
15	Arumugam	39	3	14.5	+	-	-	26	9.8	3.2	0.67	8.9	3.5	0.6	7.5	2.8
16	Narayanan	29	3.5	19.6	+	+	+	25	7.7	3	0.61	9.7	3.6	0.63	8.2	2.9
17	Joseph	30	2.5	18	+	+	+	23	7.5	2.9	0.61	9.5	3.4	0.64	5.9	2
18	Chidambaram	35	3	19.2	+	+	+	25	6.9	2.6	0.63	9.1	3	0.67	7.2	2.7
19	Peter	29	2.5	18.8	+	+	+	24	5.6	2.1	0.62	9.1	3.4	0.63	5.7	2.1
20	Diwakaran	25	2.5	18.6	+	+	+	25	6.1	1.9	0.68	7.1	2.4	0.66	5.4	1.9
21	Hariharan	28	3	18.5	+	+	+	26	7.5	2.8	0.62	6.1	2.1	0.66	7.5	2.9
22	Sundarram	30	2.5	19.5	+	+	+	23	5.9	2.2	0.63	7.5	2.9	0.62	9.1	3.3
23	Raj vijay	25	2	13.5	+	-	-	25	8.9	3.5	0.61	6.7	2.5	0.63	8.1	2.9
24	Samuel	27	2.5	16.8	+	+	+	26	8.5	3.5	0.59	9.9	3.9	0.61	8.5	6.4
25	Kowshik	27	3	16.5	+	+	+	27	6.3	2	0.68	7.3	2.8	0.61	7.9	2.8
26	Geethanesan	32	3	16.8	+	+	+	25	5.2	2.6	0.63	5.4	1.9	0.63	7.5	2.9
27	Sivakumar	29	2	17	+	+	+	27	8.5	3.5	0.59	9.7	3.8	0.61	9.2	3.5

S NO	Name	Age	VOLUME	SPERM	MOT A	MOT A+B	BOTH	MORPHO	RU PSV	RU EDV	RU RI	RM PSV	RM EDV	RM RI	RL PSV	RL EDV
28	Chidambaranadan	40	2.5	18.8	+	-	-	24	6.1	2.4	0.61	9.5	3.8	0.6	5.7	2.2
29	Prasannaraj	28	3	12.9	+	+	+	26	6.2	2.4	0.67	9.6	3.6	0.62	5.8	2.3
30	Gunalayan	34	2.5	19.5	+	+	+	24	5.6	3.3	0.61	9.9	3.7	0.63	5.9	2.2
31	Durairaj	30	2.54	17.5	+	+	+	25	7.4	2.3	0.68	5.3	2	0.61	8.3	3.4
32	Solomon	29	2	10.9	+	+	+	26	9.1	3.5	0.61	10.1	3.7	0.63	5.1	2.1
33	Anandhan	31	3	12.9	+	+	+	27	7.3	2.7	0.63	8.7	3.7	0.58	5.9	2.2
34	ArumugaRajan	35	2.5	10.8	+	+	+	27	6.5	2.5	0.62	9.1	3.7	0.59	6.1	2.1
35	Paramasivan	29	2.5	19.8	+	+	+	26	7.9	3.1	0.61	6.5	2.5	0.61	5.9	2.2
36	Sarathkumar	23	2	18.5	+	+	+	27	9.8	3.9	0.6	5.4	1.7	0.68	8.3	3.7
37	Sasikumar	33	3	18.5	+	+	+	25	7.5	2.8	0.62	6.8	2.5	0.63	8.4	3.5
38	Rahman	29	2.5	14.9	+	+	+	24	9.1	3.7	0.59	6.3	2.3	0.62	7.2	2.8
39	Aashiq	25	3	17.5	+	+	+	22	10.5	4.1	0.61	7.5	3.2	0.58	8.6	3
40	Shanmuganathan	39	3	17.6	+	+	+	28	9	3.5	0.61	5.8	2.5	0.57	6.9	2.4
41	Aravindhan	27	3	18.8	+	+	+	25	6.3	2.5	0.6	5.1	2.1	0.59	5.9	2.2
42	Berin Raj	38	2	15.6	+	+	+	23	8.3	3	0.63	6.7	2.6	0.61	7.7	2.5
43	Baskaran	30	2.5	15.8	+	+	+	24	9.1	3.6	0.6	9.9	3.8	0.62	5.3	2.1
44	Divyan	32	3	15.8	+	+	+	27	9.1	3.7	0.59	6.1	1.9	0.68	7.5	2.8
45	Deepakumar	31	3	16	+	+	+	24	9.5	3.8	0.6	7.1	2.4	0.66	6.1	2.1
46	Goutham Rajan	31	2.5	17.8	+	-	-	25	6.9	2.8	0.59	9.7	3.8	0.6	5.1	2.1
47	Mallayan	39	3	17.5	+	+	+	27	5.1	2.1	0.59	10.7	4.2	0.61	6.7	2.5
48	Iniyan	27	3	16.8	+	+	+	22	8.5	3.5	0.59	6.3	2	0.68	8.5	3.5
49	Mohamed haji	34	3	11.7	+	+	+	25	6.9	2.6	0.62	7.2	3	0.59	8.3	3.2
50	Vadivel	w	4	14.9	+	+	+	27	9.2	3.7	0.6	8.4	3.1	0.63	6.8	2.4

RL RI	LU PSV	LU EDV	LU RI	LM PSV	LM EDV	LM RI	LL PSV	LL EDV	LL RI	RI	R TESTIS V	L TESTIS V	SR.TST	S FSH	S LH	PROLACTIN
0.61	7.6	2.8	0.62	7.8	2.9	0.59	6.3	2.2	0.64	0.62	17.2	18.5	5.5	3.9	5.2	7.3
0.59	7.6	2.8	0.62	8.5	3.4	0.6	8.1	2.9	0.63	0.61	18.1	17.9	7.5	4.9	6.1	8.5
0.62	5.7	2.2	0.62	5.8	2.3	0.61	5.3	1.9	0.65	0.63	18	18.5	3.2	6.4	5.6	5.8
0.61	9.5	3.8	0.6	9.6	3.6	0.62	9.7	3.6	0.63	0.61	18.65	18.05	4.9	7.7	5.1	10.5
0.61	8.6	3	0.65	6.9	2.4	0.65	5.9	2.4	0.69	0.63	17	19	3.2	5.7	5.7	10.5
0.66	7.3	2.7	0.63	5.8	2.3	0.59	6.6	2.4	0.63	0.63	18	17.6	5	5.5	5.6	9.7
0.59	6.8	2.7	0.61	8.5	2.9	0.66	9.5	3.6	0.62	0.62	18.2	18.5	5	7.4	7	4.79
0.63	6.5	2.5	0.62	7.9	3.1	0.61	5.3	2	0.61	0.63	17.5	18	3.2	6.5	6.2	6.02
0.58	9.1	3.7	0.59	6.5	2.5	0.61	5.4	2.3	0.58	0.6	17.7	18.5	2.8	4.7	9.7	5.2
0.62	7.5	3.2	0.58	5.8	2.5	0.57	6.7	2.6	0.61	0.61	17.3	18.2	3.8	7.7	5.1	8.1
0.63	9.1	3.7	0.59	10.5	4.1	0.61	9	3.5	0.61	0.61	18.75	18.25	7.2	4.7	4.5	9.1
0.65	7.2	2.8	0.6	6.3	2.5	0.61	6.1	2.4	0.6	0.61	18	18.5	4.9	6.6	5.7	3.8
0.66	9.2	3.5	0.61	8.5	3.1	0.63	8.3	3.1	0.62	0.64	17.9	18.5	6	3.3	8.5	4.2
0.6	5.7	2.2	0.61	7.2	2.6	0.63	9.3	3.7	0.6	0.6	18	18.4	5.5	11.2	8.5	3.7
0.62	7.2	2.9	0.59	6.4	2.6	0.6	5.6	2.1	0.64	0.62	17.5	18.5	5.5	3.5	7	6.8
0.64	9.9	3.7	0.63	5.9	2.2	0.63	5.6	2.1	0.62	0.63	18.6	18.5	5.7	10.1	6.9	5.1
0.66	7.8	3	0.61	7.5	2.4	0.67	8.8	2.8	0.68	0.64	18.7	17.7	7.2	11.9	10.5	11.6
0.62	7.9	2.9	0.63	9.8	3.8	0.61	6.9	2.5	0.64	0.63	18	17.9	5.3	6.8	5.4	13.3
0.64	7.7	2.8	0.64	7.4	2.9	0.61	6.3	2.4	0.62	0.62	17.7	18.4	6.1	5.8	5.9	10.9
0.63	7.7	3	0.67	6.9	2.8	0.69	5.1	1.9	0.63	0.65	18.3	18.5	4.7	6.2	6	5.2
0.61	8.7	3	0.65	9.7	3.8	0.6	5.1	2.1	0.59	0.62	18.1	17	5.9	8.8	5.3	4.4
0.64	5.1	2.1	0.59	6.1	2.5	0.59	8.1	3.2	0.6	0.61	19.2	18.7	5.8	7.1	5.6	7.4
0.64	9.7	3.4	0.65	10.7	4.2	0.61	6.7	2.5	0.62	0.62	18	18.5	6.8	7.1	4.8	8.4
0.6	9.7	3.6	0.63	7.7	3	0.61	5.3	1.9	0.65	0.62	17.9	18	4.8	11.7	5.6	5.5
0.65	8.5	2.9	0.66	8.9	3.4	0.61	9.7	3.7	0.62	0.64	17.5	17.5	7.1	8.8	7.5	8.2
0.61	9.1	3.3	0.64	8.1	2.9	0.64	9.3	3.7	0.6	0.63	17.5	18.2	6.2	11.4	5.9	5.1
0.62	9.3	3.4	0.63	5.6	2.3	0.59	5.9	2.1	0.64	0.61	18	18.6	5.9	10.5	6.8	8.4

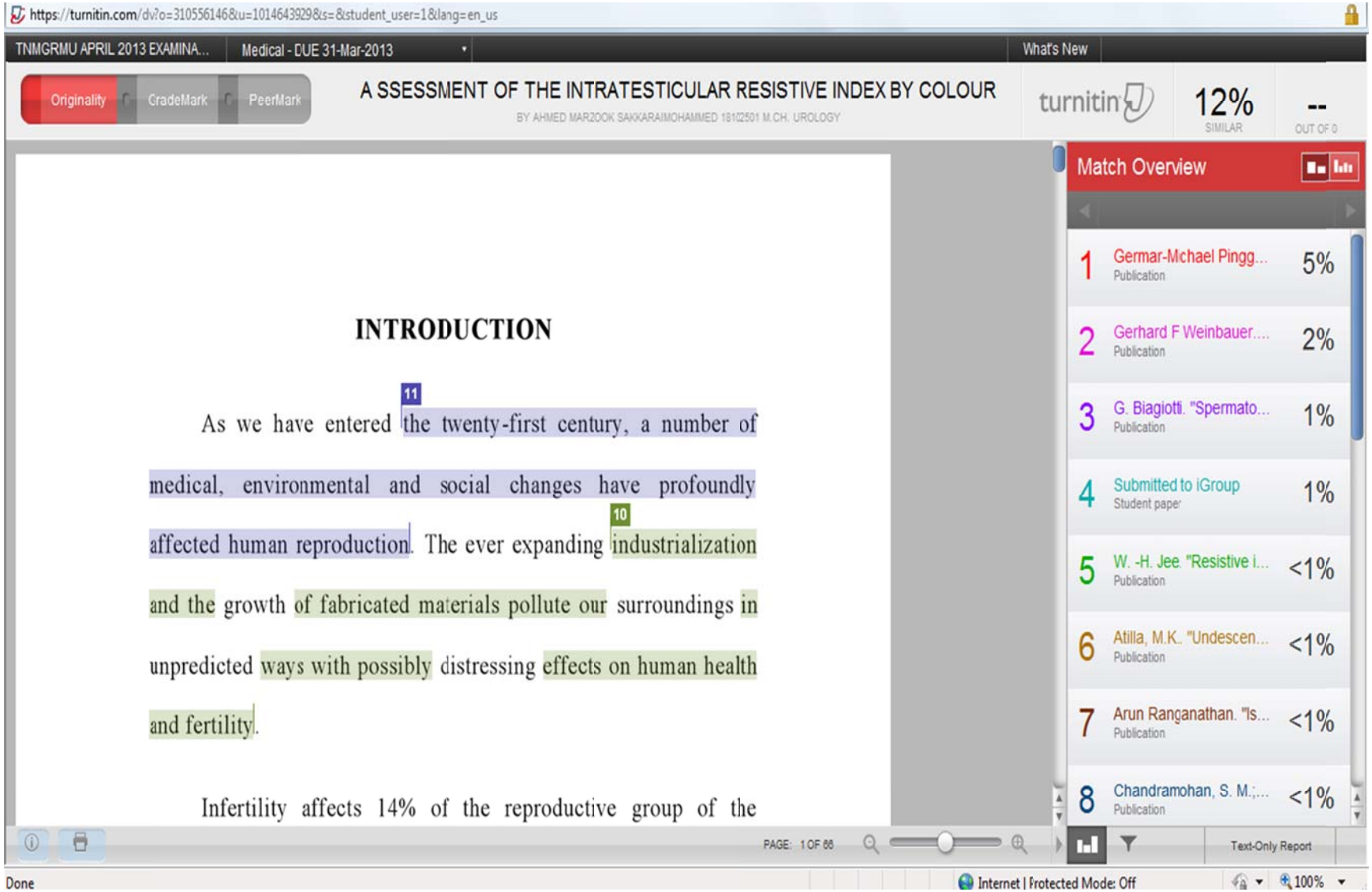
RL RI	LU PSV	LU EDV	LU RI	LM PSV	LM EDV	LM RI	LL PSV	LL EDV	LL RI	RI	R TESTIS V	L TESTIS V	SR.TST	S FSH	S LH	PROLACTIN
0.62	6.3	2.6	0.59	5.8	2.1	6.3	8.2	3.3	0.6	0.6	19	18.5	5.2	4.8	5.9	11.9
0.67	6	2.5	0.59	9.4	3.6	0.61	9.2	3.6	0.61	0.65	18.75	17.75	4.7	11.4	7.5	4.9
0.63	6.1	2.4	0.6	8.2	3	0.64	6.5	2.5	0.61	0.62	18.5	18	5.2	8.5	5.9	9.5
0.59	5.4	2.3	0.58	6.3	2.5	0.6	6.9	2.6	0.61	0.61	18	17.5	5.09	7.2	6.6	7.8
0.59	8.3	3	0.63	10.3	4	0.61	7.1	2.4	0.66	0.62	18	18.5	4.5	6.6	5.6	5.06
0.62	6.7	2.6	0.61	7.7	2.4	0.69	9.1	3.6	0.6	0.63	18	17.5	5.1	12.4	5.5	4.06
0.66	7.7	2.5	0.67	9.9	3.8	0.62	7.7	3.1	0.58	0.63	18	17.7	4.5	10.3	5.4	6.9
0.63	7.5	2.9	0.62	5.3	2.1	0.6	8.9	3.2	0.64	0.61	18.5	18.2	2.9	7.7	5.6	11.2
0.55	6.7	2.6	0.61	8.2	2.7	0.66	5.9	2.4	0.59	0.61	17.7	18	3	4.8	5	14.1
0.58	4.9	1.9	0.61	5.8	2.3	0.59	6.3	2.1	0.66	0.61	18	18.5	5	6.8	5.2	8.5
0.61	8.5	2.8	0.66	6.6	2.4	0.63	5.4	2.1	0.61	0.62	17.6	18.9	4.5	7.5	5.1	14.1
0.65	5.9	2	0.66	6.5	2.6	0.59	9.5	3.6	0.62	0.61	18.7	18.3	6.9	7.5	5.7	11.2
0.65	7.3	2.7	0.63	6.8	2.7	0.61	8.5	2.9	0.66	0.62	18	18	3.5	7.8	6	7.8
0.62	6.1	2.1	0.66	5.9	2.2	0.63	6.9	2.6	0.62	0.62	18.5	19.5	8	7.8	5.4	8.1
0.67	7.5	2.9	0.62	10.3	4	0.61	7.7	2.4	0.69	0.64	18.3	17.7	5.5	6.8	6.8	13.3
0.6	7.1	2.4	0.66	7.3	3.1	0.58	8.9	3.2	0.64	0.62	18.7	18.3	5.5	5.9	5.9	3.7
0.62	5.9	2.2	0.63	8.9	3.5	0.61	9.4	3.9	0.58	0.62	18.2	18.3	4.5	6.8	5	5.2
0.66	7.5	2.9	0.62	6.7	2.5	0.63	5.7	2.2	0.61	0.63	19.1	18.4	5.5	12.4	5.6	10.6
0.59	9.7	3.4	0.65	5.1	1.9	0.63	5.1	2.1	0.59	0.6	19	18	5.1	7.7	4.8	14.1
0.62	8.1	3.2	0.6	6.1	2.5	0.59	9.1	3.3	0.64	0.61	19.1	18.9	4.9	9.2	6.8	6.2
0.59	6.1	2.4	0.61	6.2	2.4	0.61	9.7	3.6	0.63	0.62	18.35	19.15	5.2	10.1	5.5	9.3
0.62	6.8	2.5	0.63	7.5	3	0.59	8.2	3.1	0.62	0.61	19.1	18.9	3.9	5.9	4.9	8.4
0.64	6.2	2.5	0.59	7.2	2.8	0.61	6.8	2.5	0.62	0.61	19.4	17.9	4.7	11.5	6.6	5.26

S NO	Name	Age	VOLUME	SPERM	MOT A	MOT A+B	BOTH	MORPHO	RU PSV	RU EDV	RU RI	RM PSV	RM EDV	RM RI	RL PSV	RL EDV
1	Prashanth	25	3	125	-	-	-	60	5.4	2.8	0.48	6.5	3.3	0.5	5.9	2.8
2	Priyan	22	3.5	93	-	-	-	56	7.5	3.7	0.51	5.1	2.6	0.48	8.9	4.3
3	Prabhakaran	24	4	110	-	-	-	48	5.8	3	0.48	6.3	3	0.51	5.9	2.7
4	Gururajan	23	4	100	-	-	-	52	8.5	3.5	0.59	5.8	3	0.48	6.3	3
5	Irudiyaraj	22	3.5	115	-	-	-	50	6.5	3.2	0.51	6.8	2.8	0.56	6	2.7
6	Gunasekar	40	2	135	-	-	-	45	10.1	4.7	0.53	9.8	4.8	0.52	9.5	4.1
7	Muthukumaran	35	3	90	-	-	-	43	5.4	2.7	0.49	5.6	2.6	0.53	5.9	2.9
8	Santhosh	25	2.5	85	-	-	-	56	10.5	5.3	0.49	6.9	3.5	0.5	8.2	3.9
9	Satiyaprabhat	34	4	60	-	-	-	45	6.5	3.4	0.47	8.2	4	0.51	7.1	3.3
10	Santhosh sivam	30	3.5	74	-	-	-	45	5.8	2.6	0.55	6.9	3.4	0.51	6.8	2.9
11	Prabhushankar	27	3	65	-	-	-	40	8.6	4.1	0.52	5.9	2.8	0.51	7.2	3.2
12	Kartikeyan	29	4	80	-	-	-	54	9.8	4.1	0.58	5.1	2.3	0.54	5.6	2.7
13	Balamurali	35	3	92	-	-	-	46	8.6	3.6	0.58	8.3	4.2	0.49	6.1	2.9
14	Ramesh	26	4	110	-	-	-	42	5.9	2.8	0.52	6.2	2.8	0.55	9.5	3.9
15	Kathiravan	35	3.5	70	-	-	-	52	6.1	2.9	0.51	7.2	3.7	0.48	6.9	3.1
16	Balsubramanaian	34	3	64	-	-	-	48	8.9	4.4	0.5	8	3.5	0.56	7.8	3.9
17	Praveen	22	3.5	85	-	-	-	49	7.3	3.4	0.54	6.9	2.9	0.57	6.1	2.5
18	karthik	28	2.5	74.5	-	-	-	54	7.8	4.1	0.47	9.3	4.9	0.47	6.2	3.5
19	Praveen das	26	4	120	-	-	-	55	5.7	2.9	0.5	7.1	3.8	0.46	5.4	2.7
20	Muniswaran	37	3.5	72	-	-	-	53	7.3	3.6	0.5	7.9	3.7	0.53	8.2	4.3
21	Karthik rajan	27	2.5	95	-	-	-	45	9.12	4.7	0.48	10	5	0.5	8.6	3.8
22	Prathap	28	3.5	80.5	-	-	-	44	7.1	3.6	0.5	5	2.4	0.51	5.9	2.7
23	Subramani	40	3	70.5	-	-	-	41	5.7	2.4	0.58	7.1	3.8	0.46	5.4	2.7
24	kamalan	30	2.5	74.5	-	-	-	50	8.8	4.1	0.53	9.3	3.9	0.58	6.2	2.5
25	Sathish	26	3.5	95.3	-	-	-	44	7.8	3.9	0.5	8.3	3.8	0.54	8.2	3.9
26	Rameshkumar	28	3.5	64.2	-	-	-	47	8.9	4.1	0.53	8.6	3.6	0.58	7.8	3.9
27	Kumaravelan	27	3.5	72.8	-	-	-	49	7.2	3.2	0.56	6.9	3.2	0.54	6.5	2.9
28	Ramkumar	39	3.5	72.8	-	-	-	54	7.4	3.8	0.48	6	2.9	0.51	7.1	3.6
29	Babu	30	3	92.6	-	-	-	45	9.6	4.5	0.53	6.8	3.6	0.46	7	3.5
30	Arulkumar	29	3.5	110	-	-	-	44	6.7	3.9	0.41	7.5	3.6	0.52	6.4	3.7
31	Kamalakkannan	29	3.5	73.6	-	-	-	43	7.8	3.6	0.53	5.7	2.8	0.5	9.2	4.8
32	Joshua seelan	35	3.5	71.5	-	-	-	39	5.1	2.5	0.51	7.3	3.4	0.54	8.2	3.8

S NO	Name	Age	VOLUME	SPERM	MOT A	MOT A+B	BOTH	MORPHO	RU PSV	RU EDV	RU RI	RM PSV	RM EDV	RM RI	RL PSV	RL EDV
33	Rahamatullah	32	3	71	-	-	-	40	8.2	3.9	0.52	7.6	3.6	0.53	8.8	4.4
34	Alex raj	31	4	73.3	-	-	-	49	5.2	2.8	0.46	7.6	3.6	0.53	8.1	3.8
35	sairam	34	3	62	-	-	-	44	8.2	3.8	0.54	5.4	2.8	0.48	7.2	3.5
36	Antony	23	3.5	93	-	-	-	58	7.4	3.8	0.48	8.8	4.4	0.5	5.3	2.4
37	Nikil	35	3.5	73	-	-	-	45	5.1	2.5	0.52	5.4	2.8	0.48	9.1	4.5
38	Jose	29	3.8	95	-	-	-	45	5.5	2.8	0.49	7.6	3.8	0.5	6.2	3.1
39	Thirumani	25	5	120	-	-	-	56	9.8	4.8	0.51	7.6	3.5	0.53	8.2	4.1
40	Sanjjeev	32	3	70	-	-	-	39	9.2	3.8	0.58	8.4	3.9	0.53	5.3	2.4
41	Selvakumar	34	3.5	72.8	-	-	-	40	9.8	4.7	0.55	6.8	3.9	0.43	8.1	3.7
42	Guru	30	2.8	74.2	-	-	-	41	5.9	3.1	0.48	7.5	3.5	0.53	6.7	3.2
43	Muthuselvam	33	3	94.5	-	-	-	45	6.5	3.4	0.47	8.2	4	0.51	7.1	3.3
44	Arumugaraj	29	2.5	82	-	-	-	48	10.5	5.3	0.49	8.2	3.9	0.53	6.9	3.5
45	Lucas kevin	28	3.2	72.6	-	-	-	46	5.9	2.9	0.5	5.6	2.6	0.53	5.4	2.7
46	Prakash Raj	34	3.8	75	-	-	-	46	9.7	4.8	0.5	10.7	5.5	0.48	6.7	3.8
47	Sivakumar	32	2.5	90	-	-	-	45	5.8	2.6	0.54	6.5	3.1	0.52	8.5	4.4
48	Suresh paulraj	28	3	63.5	-	-	-	44	7.2	3.6	0.5	6.5	3.9	0.41	6.9	3.2
49	Amanullah	37	3.5	64	-	-	-	43	8.9	4.1	0.53	8.6	3.6	0.58	7.8	3.9
50	Logathan	29	3.5	72.8	-	-	-	49	7.2	3.2	0.56	6.9	3.2	0.54	6.5	2.9

RL RI	LU PSV	LU EDV	LU RI	LM PSV	LM EDV	LM RI	LL PSV	LL EDV	LL RI	RI	R TESTIS V	L TESTIS V	SR.TST	S FSH	S LH	PROLACTIN
0.51	7.2	3.2	0.56	6.9	3.2	0.54	6.51	2.9	0.51	0.51	18	19.5	7.9	11.2	8.4	5.7
0.52	9.7	4.8	0.5	10.7	5.5	0.48	6.7	2.8	0.58	0.51	18.2	18.5	3.7	2.1	4.8	6.7
0.53	5.8	2.6	0.54	6.5	3.1	0.52	8.5	4.1	0.5	0.52	18	18.5	2.9	1.8	2.6	4.1
0.51	5.8	2.6	0.54	9.1	4.1	0.55	7.3	3.6	0.5	0.53	19	18	2.8	8.2	5.9	14.3
0.54	7.2	3.6	0.5	7	3.3	6.9	3.1	0.55	0.52	0.53	18.5	17.8	4.7	8.2	7.9	9.5
0.56	9	4.4	0.51	8.9	4.5	0.49	9.3	4.6	0.5	0.52	18.5	19	6.7	2.4	5.9	3.9
0.5	5.8	2.5	0.56	6.1	2.9	0.51	6.6	3.3	0.5	0.51	18	18.3	2.9	1.7	8.4	4.3
0.53	5.6	2.5	0.54	7.3	3.5	0.51	9	4.6	0.53	0.52	17.5	18	6.3	5.6	8.8	6.06
0.53	5.9	2.6	0.56	9.6	4.9	0.49	6.3	3	0.52	0.51	18	19	3.2	2.8	3.6	5.6
0.56	5.9	3.1	0.48	7.5	3.5	0.53	6.7	3.2	0.51	0.52	18	19.5	7.9	10.1	7.8	10.6
0.56	9.5	4.1	0.57	6.8	2.9	0.58	8.1	3.7	0.54	0.54	19	18.5	2.9	6.2	5.4	6.02
0.51	8.9	3.7	0.58	9.5	3.4	0.57	8.3	3.5	0.57	0.56	18	19.5	3.7	5.9	7.2	3.6
0.52	7.3	3.4	0.53	9.1	4.4	0.51	6.2	2.7	0.56	0.53	18.1	17	3.6	4.8	3	6.06
0.58	8.1	4.6	0.57	5.8	2.9	0.5	8.2	3.8	0.53	0.54	19	19.5	2.8	1.5	1.7	4.4
0.54	8.2	3.3	0.59	8.1	4	0.5	7.9	3.7	0.52	0.52	18.5	18	7.5	11.2	3.4	5.1
0.49	8.1	3.9	0.51	5.4	2.6	0.52	5.2	2.1	0.58	0.53	18.5	18	6.6	3.2	4.9	11.5
0.59	7.8	3.2	0.59	8.3	3.6	0.56	9.2	3.9	0.58	0.56	18	19	4.7	9.9	7.2	5.1
0.49	5.6	3.3	0.41	7.1	3.9	0.45	7.5	3.8	0.49	0.45	18	18.5	7.9	12.4	8.6	5.2
0.5	7.5	3.9	0.48	6.1	3.6	0.41	8	3.8	0.52	0.48	20	18	6.2	10.4	7.1	12.8
0.48	7.1	3.6	0.49	6.6	3.4	0.48	5.9	2.9	0.5	0.49	18	19	4.3	11.7	8.7	5
0.55	8.8	3.9	0.56	6.5	2.9	0.54	6.6	3.2	0.52	0.52	18.5	19	5.1	6.5	7.3	8.7
0.54	7.3	3.6	0.5	7.9	3.7	0.53	8.2	4.3	0.48	0.5	17.9	18.6	4.3	4.7	8	8.7
0.5	7.5	3.9	0.48	6.1	3.6	0.41	8	3.8	0.52	0.47	19.1	18	6.2	10.4	7.1	8.8
0.59	5.6	3.3	0.61	7.1	2.9	0.58	7.2	3.8	0.5	0.56	18	18.5	7.9	12.4	8.6	15.2
0.52	8.1	3.9	0.51	5.4	2.6	0.52	5.2	2.7	0.48	0.5	18	17	4.7	9.9	7.2	5.1
0.49	7.3	3.9	0.46	6.9	3.9	0.43	6.1	3.8	0.44	0.48	18.5	17	6.7	3.2	4.9	8.5
0.51	8.2	3.3	0.59	8.1	4	0.5	7.9	3.7	0.52	0.53	18	19.5	8	11.3	8.1	5.8
0.49	8.9	4.4	0.5	6.8	3.9	0.42	9.3	4.5	0.51	0.48	18	17.7	5	2	2.3	5.2
0.5	6.9	3.5	0.5	5.2	2.5	0.52	6.2	2.9	0.53	0.5	18	18.5	5	2.3	2.5	5.2
0.42	9	3.9	0.56	8.3	4.2	0.5	8.2	3.8	0.53	0.49	18	18.5	2.9	1.8	2.6	4.1
0.47	8.6	4.9	0.47	5.9	3.4	0.42	6.6	3.3	0.5	0.48	19	18.5	2.8	1.5	1.7	4.7
0.53	9.8	4.9	0.5	5.3	2.8	0.47	8.4	4.3	0.49	0.5	17.3	18.2	6.1	7	3.2	8.5

RL RI	LU PSV	LU EDV	LU RI	LM PSV	LM EDV	LM RI	LL PSV	LL EDV	LL RI	RI	R TESTIS V	L TESTIS V	SR.TST	S FSH	S LH	PROLACTIN
0.5	7.4	3.7	0.5	9.8	4.9	0.5	8	3.8	0.52	0.5	18.4	19.2	6	7	3.2	8.5
0.53	6.7	3.4	0.49	8.5	4.2	0.51	7.3	3.2	0.56	0.52	19.3	18.4	3.7	4.9	3.1	6.7
0.51	6.9	3.5	0.49	8.2	3.9	0.52	7.6	3.8	0.5	0.5	18.5	17.5	5.5	9.5	3.6	6.08
0.55	7.8	3.8	0.51	6.9	3.8	0.44	7.5	3.2	0.48	0.49	18.9	18.1	5.6	9.7	3.5	6.8
0.5	5.6	2.8	0.5	8.7	4.4	0.49	5.2	2.8	0.46	0.49	19	18	7	5.6	1.7	8.2
0.5	5.9	2.8	0.52	9.3	4.4	0.53	6.8	3.6	0.47	0.5	18.5	17.5	3.7	5.9	7.2	8.6
0.5	9	4.4	0.5	8.6	4.6	0.46	7.6	3.8	0.5	0.5	19	18	7	5.6	1.7	8.02
0.54	7.8	3.5	0.55	8.4	3.6	0.56	9.2	4	0.57	0.55	18.5	19	5.5	4	8	11.2
0.52	8.2	3.9	0.52	9	4.9	0.45	8	3.8	0.52	0.5	18.7	18.8	2.8	6.3	5.5	6.04
0.51	5.9	2.6	0.56	9.6	4.9	0.49	6.3	3	0.52	0.51	18.3	19.5	8.1	10.2	7.9	10.3
0.53	5.8	2.9	0.5	6.9	3.4	0.51	6.8	3.4	0.5	0.5	18	19	3.2	2.2	4.6	7
0.5	9.6	4.8	0.5	7.3	3.5	0.51	5.6	2.8	0.5	0.5	17.5	18	7.3	6.6	9.8	7.6
0.5	6.1	2.9	0.51	6.6	3.3	0.5	6.8	3.5	0.48	0.49	19.2	18.1	2.8	1.8	8.3	10.2
0.43	7.5	3.7	0.51	5.1	2.6	0.48	8.9	4.3	0.52	0.48	18	19	4.7	3.1	5.8	7.7
0.48	5.8	3	0.48	5.9	2.7	0.53	6.3	3.6	0.42	0.49	18.5	18	2.7	1.8	2.7	4.1
0.54	6.5	3.3	0.5	5.9	2.8	0.51	5.4	2.8	0.48	0.47	18	19.9	7.7	12.1	8.7	5.9
0.49	7.3	3.9	0.46	6.9	3.9	0.43	6.7	3.8	0.43	0.48	18.5	18	6.7	3.5	4.9	8.5
0.51	8.2	3.3	0.59	8.1	4	0.5	7.9	3.7	0.52	0.53	17.2	18.5	7.2	11.5	3.4	5.1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As we have entered the twenty-first century, a number of medical, environmental and social changes have profoundly affected human reproduction. The ever expanding industrialization and the growth of fabricated materials pollute our surroundings in unpredicted ways with possibly distressing effects on human health and fertility.
Infertility affects 14% of the reproductive group of the
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